DOCUMENT-IDENTIFIER: US 20010036943 A1
TITLE: Pharmaceutical composition for treatment of acute, chronic pain and/or neuropathic pain and migraines

Abstract Paragraph:
Pharmaceutical compositions are disclosed for the treatment of acute, chronic and/or neuropathic pain. The pharmaceutical compositions are comprised of a therapeutically effective combination of a nicotine receptor partial agonist and an analgesic agent and a pharmaceutically acceptable carrier. The analgesic agent is selected from opioid analgesics, NMDA antagonists, substance P antagonists, COX 1 and COX 2 inhibitors , tricyclic antidepressants (TCA), selective serotonin reuptake inhibitors (SSRI), capsaicin receptor agonists, anesthetic agents, benzodiazepines, skeletal muscle relaxants, migraine therapeutic agents, anti-convulsants, anti-hypertensives, anti-arrythmics, antihistamines, steroids, caffeine, and botulinum toxin. The method of using these compounds and a method of treating acute, chronic and/or neuropathic pain and migraine in a mammal including a human is also disclosed.

CLAIMS:

2. The pharmaceutical composition according to claim 1, wherein said analgesic agent is selected from opioid analgesics, NMDA antagonists, substance P antagonists, COX 1 and COX 2 inhibitors , tricyclic antidepressants (TCA), selective serotonin reuptake inhibitors (SSRI), capsaicin receptor agonists, anesthetic agents, benzodiazepines, skeletal muscle relaxants, migraine therapeutic agents, anti-convulsants, anti-hypertensives, anti-arrythmics, antihistamines, steroids, caffeine, N-type calcium channel antagonists, and botulinum toxin.

18. The method according to claim 17 wherein the analgesics are selected from opioid analgesics, NMDA antagonists, substance P antagonists, COX 1 and COX 2 inhibitors, tricyclic antidepressants (TCA), selective serotonin reuptake inhibitors (SSRI), capsaicin receptor agonists, anesthetic agents, benzodiazepines, skeletal muscle relaxants, migraine therapeutic agents, anti-convulsants, anti-hypertensives, anti-arrythmics, antihistamines, steroids, caffeine, N-type calcium channel antoginists and botulinum toxin or their pharmaceutically acceptable salt or optical isomers.

33. A pharmaceutical composition for treating a disorder or condition selected from the group consisting of diseases and conditions in which pain predominates, including acute pain, chronic pain, neuropathic pain and migraine, and including soft tissue and peripheral damage, such as acute trauma, osteoarthritis, rheumatoid arthritis, musculo-skeletal pain, particularly after trauma, spinal pain, dental pain, myofascial pain syndromes, headache, episiotomy pain, and burns; deep and visceral pain, such as heart pain, muscle pain, eye pain, orofacial pain, for example, odontalgia, abdominal pain, gynaecological pain, for example, dysmenorrhea, and labor pain; pain associated with nerve and root damage, such as pain associated with peripheral nerve disorders, for example, nerve entrapment and brachial plexus avulsions, amputation, peripheral neuropathies, tic douloureux, atypical facial pain, nerve root damage, and arachnoiditis; pain associated with carcinoma, often referred to as cancer pain; central nervous system pain, such as pain due to spinal cord or brain stem damage; low back pain; sciatica; headache, including migraine, acute or chronic tension headache, cluster headache, temporomandibular pain and maxillary sinus pain; ankylosing spondylitis, gout; post operative pain; and scar pain, in a mammal, including a human, the method comprising administering to said mammal respectively a pain attenuating effective amount of a pharmaceutical composition comprising: (a) a nicotine receptor partial agonist or a pharmaceutically acceptable salt thereof; (b) an analgesic agent or a pharmaceutically acceptable salt thereof and (c) a pharmaceutically acceptable carrier, wherein the active agents "a" and "b" above are present in amounts that render the composition effective in treating acute, chronic and/or neuropathic pain and migraine.

34. A method of treating a disorder or condition selected from the group consisting of diseases and conditions in which pain predominates, including acute pain, chronic pain, neuropathic pain and migraine, and including soft tissue and peripheral damage, such as acute trauma, osteoarthritis, rheumatoid arthritis, musculo-skeletal pain, particularly after trauma, spinal pain, dental pain, myofascial pain syndromes, headache, episiotomy pain, and burns; deep and visceral pain, such as heart pain, muscle pain, eye pain, orofacial pain, for example, odontalgia, abdominal pain, gynaecological pain, for example, dysmenorrhea, and labor pain; pain associated with nerve and root damage, such as pain associated with peripheral nerve disorders, for example, nerve entrapment and brachial plexus avulsions, amputation, peripheral neuropathies, tic douloureux, atypical facial pain, nerve root damage, and arachnoiditis; pain associated with carcinoma, often referred to as cancer pain; central nervous system pain, such as pain due to spinal cord or brain stem damage; low back pain; sciatica; headache, including migraine, acute or chronic tension headache, cluster headache, temporomandibular pain and maxillary sinus pain; ankylosing spondylitis, gout; post operative pain; and scar pain, in a mammal, including a human, the method comprising administering to said mammal respectively a pain attenuating effective amount of a pharmaceutical composition comprising: (a) a nicotine receptor partial agonist or a pharmaceutically acceptable salt thereof; (b) an analgesic agent or a pharmaceutically acceptable salt thereof and (c) a pharmaceutically acceptable carrier, wherein the active agents "a" and "b" above are present in amounts that render the composition effective in treating acute, chronic and/or neuropathic pain and migraine.

applied

DOCUMENT-IDENTIFIER: US 5670484 A
TITLE: Method for treatment of skin lesions associated with cutaneous cell-proliferative disorders

Abstract Text (1):
The invention is a method for treatment of cutaneous cell-proliferative disorders. More specifically, the method of the invention is useful in mitigating and inducing remission of lesions associated with such disorders and in controlling related symptoms of the disorders (such as scaling and itching). According to the method of the invention, an invertebrate neurotoxin is administered to the skin of the host at or near the site of a lesion. The preferred neurotoxin for use in the method of the invention is Botulinum toxin, particularly Botulinum toxin A.

CLAIMS:

1. A method for mitigating or inducing remission of a skin lesion associated with a cutaneous cell-proliferative disorder in a mammal comprising administering a therapeutically effective amount of a Botulinum toxin in a pharmaceutically safe form to the mammal by delivery of the Botulinum toxin to the site of the lesion.

2. The method according to claim 1 wherein the Botulinum toxin is administered by subcutaneous injection.

3. The method according to claim 1 wherein the Botulinum toxin is Botulinum toxin A.

4. A method for controlling symptoms associated with the onset or presence of a cutaneous cell-proliferative disorder in a mammal comprising administering a therapeutically effective amount of a Botulinum toxin in a pharmaceutically safe form to the mammal by delivery of the Botulinum toxin to the mammal's skin.

5. A method for mitigating or inducing remission of a skin lesion associated with a cutaneous cell-proliferative disorder in a mammal comprising administering a therapeutically effective amount of a Tetanus toxin in a pharmaceutically safe form to the mammal by delivery of the Tetanus toxin to the site of the lesion.

7. A method for mitigating or inducing remission of a skin lesion associated with a cutaneous cell-proliferative disorder in a mammal comprising administering a therapeutically effective amount of a biologically active fragment of a Tetanus toxin in a pharmaceutically safe form to the mammal by delivery of the Tetanus toxin to the site of the lesion.

9. A method for controlling symptoms associated with the onset or presence of a cutaneous cell-proliferative disorder in a mammal comprising administering a therapeutically effective amount of a Tetanus toxin in a pharmaceutically safe form to the mammal by delivery of the Tetanus toxin to the mammal's skin.

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L1: Entry 2 of 5 File: PGPB Sep 8, 2005

DOCUMENT-IDENTIFIER: US 20050196414 A1
TITLE: Compositions and methods for topical application and transdermal delivery of botulinum toxins

CLAIMS:

131. A method according to claim 54 in which the botulinum toxin is applied topically for alteration of hair growth.

Previous Doc Next Doc Go to Doc#

prevent U.S. Pat 6,299,893
hair loss

First Hit Previous Doc Next Doc Go to Doc#

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L6: Entry 56 of 114 File: PGPB Dec 9, 2004

DOCUMENT-IDENTIFIER: US 20040248188 A1
TITLE: Methods for using tetanus toxin for benificial purposes in animals (mammals)

CLAIMS:

1. A method of modulating a neural function of an animal at a selected site affected by target neurons, the method comprising administering a therapeutically effective amount of tetanus toxin to the selected site of the animal such that the neurotoxin reversibly modulates the activity of the target neurons.

17. The method of claims 1-5, wherein the therapeutically effective amount of tetanus toxin is delivered at the selected site by injection, topical application, aerosol, or instillation into ducts or body orifices.

18. The method of claim 8, wherein the therapeutically effective amount of tetanus toxin is delivered at the selected site by injection, topical application, aerosol, or instillation into ducts or body orifices.

19. The method of claim 9, wherein the therapeutically effective amount of tetanus toxin is delivered at the selected site by injection, topical application, aerosol, or instillation into ducts or body orifices.

63. The method of claims 1, 2, 4 wherein the selected site comprises hair follicles, prostate gland, connective tissue of lax, aged skin, inflamed fibers, skin in proliferative or allergic diseases, sebaceous gland, sympathetic nerve of the circulatory system, neurons controlling an immune response in thymus, lymph nodes, or tissue having a neural immune interaction, skin, digestive tract, tonsils, anterior chamber of the eye, gastric mucosa, nasal mucosa or pterygopalatine ganglia.

84. The method of claim 78 or 81, wherein the therapeutically effective amount of tetanus toxin is delivered at the selected site by injection, topical application, aerosol, instillation into ducts or body orifices, encapsulated into liposomes or artificial vesicles with bi-layer lipid membrane.

Previous Doc Next Doc Go to Doc#

DOCUMENT-IDENTIFIER: US 20050074466 A1
TITLE: Botulinum toxin in the treatment or prevention of acne

Abstract Paragraph:
Botulinum toxin may be used to inhibit the cascade of events leading to acne. Results in preliminary studies have been dramatic. Withoout wishing to be bound by this theory, it is believed that botulinum toxin achieves this result through parasympathetic effects, inhibiting sweat gland activity, stimulating keratinocyte locomotion, anti-inflammatory effects, and possibly anti-androgenic effects. Botulinum toxin can play an important role in decreasing and even preventing the formation of acne.

CLAIMS:

1. A method for inhibiting acne vulgaris in a human who is susceptible to acne vulgaris, said method comprising delivering an effective amount of botulinum toxin A to one or more regions of the skin of the human that are susceptible to acne vulgaris.

2. A method as recited in claim 1, wherein said delivering comprises the intracutaneous injection of botulinum toxin A at multiple sites in the skin, wherein the sites of adjacent injections are separated by about 0.5 to 10 cm.

3. A method as recited in claim 1, wherein said delivering comprises the intracutaneous injection of botulinum toxin A at multiple sites in the skin, wherein the sites of adjacent injections are separated by about 1.5 to 3 cm.

4. A method as recited in claim 1, wherein said delivering comprises the topical application to susceptible regions of the skin of a composition comprising botulinum toxin A.

5. A method as recited in claim 1, wherein said delivering comprises the subcutaneous injection of the botulinum toxin A.

6. A method as recited in claim 1, wherein said delivering comprises the intramuscular injection of the botulinum toxin A.

10. A method as recited in claim 1, wherein said delivering comprises the intracutaneous injection of botulinum toxin A at multiple sites in the skin, and wherein between about 1 U and about 20 U of botulinum toxin A is injected at each site.

11. A method as recited in claim 1, wherein said delivering comprises the intracutaneous injection of botulinum toxin A at multiple sites in the skin, and wherein between about 2 U and about 3 U of botulinum toxin A is injected at each site.

12. A method as recited in claim 1, additionally comprising the inhibition of at least one rhytid by said delivering of an effective amount of botulinum toxin A to one or more regions of the skin that are susceptible to acne vulgaris and that also contain at least one rhytid.

13. A method for inhibiting acne vulgaris in a human who is susceptible to acne vulgaris, said method comprising delivering an effective amount of botulinum toxin to one or more regions of the skin of the human that are susceptible to acne vulgaris.

14. A method as recited in claim 13, wherein said delivering comprises the intracutaneous injection of

botulinum toxin at multiple sites in the skin, wherein the sites of adjacent injections are separated by about 0.5 to 10 cm.

15. A method as recited in claim 13, wherein said delivering comprises the intracutaneous injection of botulinum toxin at multiple sites in the skin, wherein the sites of adjacent injections are separated by about 1.5 to 3 cm.

16. A method as recited in claim 13, wherein said delivering comprises the topical application to susceptible regions of the skin of a composition comprising botulinum toxin.

17. A method as recited in claim 13, wherein said delivering comprises the subcutaneous injection of the botulinum toxin.

18. A method as recited in claim 13, wherein said delivering comprises the intramuscular injection of the botulinum toxin.

22. A method as recited in claim 13, additionally comprising the inhibition of at least one rhytid by said delivering of an effective amount of botulinum toxin to one or more regions of the skin that are susceptible to acne vulgaris and that also contain at least one rhytid.

23. A method as recited in claim 13, wherein the botulinum toxin comprises botulinum toxin B.

24. A method as recited in claim 13, wherein the botulinum toxin comprises botulinum toxin C.

25. A method as recited in claim 13, wherein the botulinum toxin comprises botulinum toxin D.

26. A method as recited in claim 13, wherein the botulinum toxin comprises botulinum toxin E.

27. A method as recited in claim 13, wherein the botulinum toxin comprises botulinum toxin F.

28. A method as recited in claim 13, wherein the botulinum toxin comprises botulinum toxin G.

29. A method as recited in claim 13, additionally comprising the inhibition of at least one rhytid by said delivering of an effective amount of a botulinum toxin to one or more regions of the skin that are susceptible to acne vulgaris and that also contain at least one rhytid; wherein the botulinum toxin is selected from the group consisting of botulinum toxin B, botulinum toxin C, botulinum toxin D, botulinum toxin E, botulinum toxin F, and botulinum toxin G.

Previous Doc Next Doc Go to Doc#

DOCUMENT-IDENTIFIER: US 20050123567 A1
TITLE: Botulinum toxin therapy for skin disorders

Abstract Paragraph:
Methods for treating skin disorders by local administration of a Clostridial toxin, such as a botulinum toxin, to a patient with a skin disorder.

CLAIMS:

1. A method for treating a skin disorder, the method comprising a step of administering a therapeutically effective amount of a botulinum toxin to a location of a skin disorder of a patient, wherein the skin disorder comprises a disorder selected from the group consisting of warts, corns, calluses, neuromas, ulcers, hammertoes and bunions, thereby treating the skin disorder.

2. The method of claim 1, wherein the botulinum toxin is selected from the group consisting of botulinum toxin types A, B, C, D, E, F and G.

3. The method of claim 1, wherein the botulinum toxin is a botulinum toxin type A.

4. The method of claim 1, wherein the botulinum toxin is administered in an amount of between about 1 unit and about 3,000 units.

5. The method of claim 1, wherein the administration is by topical or subcutaneous administration of the botulinum toxin.

6. A method for treating a skin disorder, the method comprising the step of locally administering between 1 unit and 3000 units of a botulinum toxin to a skin disorder of the patient, wherein the skin disorder comprises a disorder selected from the group consisting of warts, corns, calluses, neuromas, ulcers, hammertoes and bunions, thereby treating the skin disorder.

8. The method of claim 6, wherein the skin disorder is treated by reducing a pain associated with the skin disorder.

9. The method of claim 6, wherein the skin disorder is treated by reducing an inflammation associated with the skin disorder.

10. The method of claim 6, wherein the skin disorder is treated by reducing a size of the skin disorder.

11. A method for treating a wart, the method comprises the step of administering a therapeutically effective amount of a botulinum toxin to a wart, thereby treating the wart.

Previous Doc Next Doc Go to Doc#

Previous Doc Next Doc Go to Doc#
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L6: Entry 81 of 114 File: PGPB Sep 4, 2003

DOCUMENT-IDENTIFIER: US 20030166004 A1
TITLE: Endothelial-cell binding peptides for diagnosis and therapy

CLAIMS:

10. The peptide/polypeptide of claim 7, wherein the agent is selected from: alkylating agents, enzyme inhibitors, proliferation inhibitors, lytic agents, DNA or RNA synthesis inhibitors, membrane permeability modifiers, DNA intercalators, metabolites, dichloroethylsulfide derivatives, protein production inhibitors, ribosome inhibitors, inducers of apoptosis, or neurotoxins.

43. The method of claim 42, for prophylaxis or reducing the effects of a disorder selected from: hemangioma, solid tumors, leukemia, metastasis, telangiectasia, psoriasis, scleroderma, pyogenic granuloma, myocardial angiogenesis, plaque neovascularization, coronary collaterals, cerebral collaterals, arteriovenous malformations, ischemic limb angiogenesis, corneal diseases, rubeosis, neovascular glaucoma, diabetic retinopathy, retrolental fibroplasia, arthritis, diabetic neovascularization, macular degeneration, wound healing, peptic ulcer, Helicobacter related diseases, fractures, keloids, vasculogenesis, hematopoiesis, ovulation, menstruation, placentation, or cat scratch fever.

52. The method of claim 50, as part of a treatment regimen for repair of vascular damage after ischemia.

Previous Doc Next Doc Go to Doc#

DOCUMENT-IDENTIFIER: US 20050180995 A1
TITLE: Method to Improve Grafted Hair Growth And Retain Grafted Hair

CLAIMS:

2. A method for improving the growth and retention of any type of hair graft or hair transplant on the scalp of a human patient as claimed in claim 1 wherein the presynaptic neurotoxin is a botulinum toxin.

3. A method for improving the growth and retention of any type of hair graft or hair transplant on the scalp of a human patient as claimed in claim 1 wherein the presynaptic neurotoxin is botulinum toxin A.

142. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with ear disorders.

143. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with cancer.

144. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with nerve entrapment disorders.

145. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with hypercalcemia.

146. A method according to claim 51 in which the botulinum toxin comprises a fusion protein.

147. A composition according to claim 5 in which the botulinum toxin is botulinum toxin F.

148. A composition according to claim 5 in which the botulinum toxin is botulinum toxin G.

149. A method according to claim 51 in which the botulinum toxin is botulinum toxin F.

150. A method according to claim 51 in which the botulinum toxin is botulinum toxin G.

Previous Doc Next Doc Go to Doc#

WEST Search History

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DATE: Thursday, January 04, 2007

Hide?	Set Name	Query	Hit Count
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☐	L1	botulinum.clm. same hair.clm.	5
☐	L2	botulinum same hair same color	1

END OF SEARCH HISTORY

DOCUMENT-IDENTIFIER: US 20050220734 A1
TITLE: Therapy for melanin related afflictions

Summary of Invention Paragraph:
[0071] My invention includes a method for treating hair color changes a liquid shampoo-based formulation that could be topically applied to the hair follicles. It is known that certain hormones which regulate hair color through affects upon melanin release are influenced by neuropeptides the release of which, as explained below, can be influenced by a botulinum toxin.

Summary of Invention Paragraph:
[0073] Without wishing to be bound by theory a mechanism can be proposed for the efficasy of the present invenion disclosed herein. Botulinum toxin has been shown in in vitro and in vivo models to inhibit release of various neuropeptides such as cGRP, substance P and the amino acid, glutamate and acetylcholine. (Durham, 2003; Cui et al., 2002; Welch et al., 2002). These neuropeptides act as modulators influencing melanin production and thereby skin pigmentation and hair color. Additionally, a botulinum toxin has been shown to have a direct effect on the skin (Li et al.). Thus, sensory and motor nerve denervation influence the epidermal thickness in rat foot glabrous skin, including denervation following a botulinum toxinA administration.

Summary of Invention Paragraph:
[0097] My invention also encompasses use of a botulinum toxin to cause hair removal. Stress has long been associated with disturbances in hair growth and hair pigmentation. A wide range of experimental data suggests that skin nerves can indeed modulate hair follicle (HF) development, growth and/or cycling via the release of neurotransmitters, neuropeptides and/or even of neurotrophins. It is therefore conceivable that stress-induced changes in the release of these agents from perifollicular sensory and autonomic nerve fibers can alter hair growth. Most classical mediators of systemic stress responses (e.g., substance p, ACTH, CRH, prolactin, catecholamines) are now also appreciated as hair growth modulators. Moreover, the HF itself is a potent source of these stress mediators, and expresses cognate receptors for many of them. Finally, mast cells, with their `central switchboard` function in neurogenic inflammation, have recently surfaced as hair growth modulators. Stress mediators, as well as skin neuropeptides and neurotransmitters, can thus, impact hair growth indirectly via the modulation of mast cell activities. As a prominent source of neurotrophins, the hair follicle can influence its own innervation as well as neurotrophin-dependent mast cell functions. Reports of preliminary evidence that stress actually can inhibit hair growth in mice, demonstrates potential pathways by which stress may affect hair growth and color in the context of defined neuro-endocrine-immune circuits.

Detail Description Paragraph:
[0105] A 58 year-old single male can be concerned that the premature graying of his hair contributed to his ability to find a future wife, and can be seen in a dermatology clinic. Despite numerous attempts with several different hair coloring methods, the patient's hair may not change to darker color. A course of a hybrid formulation of containing heavy chains-only (100 Kd) of both botulinum toxin type-A and type-F and Ro31-8220, a potent inhibitor of protein kinase-C, admixed in a topical shampoo formulation can be decided upon. A suitable amount (1 cc/1 cm2) of the shampoo can be applied by the clinician using sterile protective gloves, along with a preventative barrier to restrict application to the patients scalp region. After two successive shampoo treatments with the same formulation spaced 4 weeks apart, a very noticeable change in hair color can be observed: from light gray to a medium black color.

CLAIMS:

8. A method for altering hair color, the method comprising the step of administering a botulinum toxin

to the hair, thereby altering the hair color.

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DATE: Thursday, January 04, 2007

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		or dysport or myobloc or neurotoxin).ti,ab,clm. and allergan.asn. and (skin or dermal or topically or topical or intradermal or melann or pigment or dermatofibromas or fibromas or dermoid or freckles or keloids or seborrheic or keratoses or actinic or albinism or melasma or vitiligo or damage).clm. and (method or process).clm.))	
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Search Results - Record(s) 101 through 114 of 114 returned.

☐ 101. 6537771. 06 Oct 00; 25 Mar 03. Use of nernstein voltage sensitive dyes in measuring transmembrane voltage. Farinas; Javier Anibal, et al. 435/29; 435/4 435/7.2 435/968. C12Q001/02 C12Q001/00 G01N033/53 .

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☐ 111. 5489510. 09 Dec 93; 06 Feb 96. Method for visual indication of cholesterol on skin surface agents used therefor and methods for producing such agents. Lopukhin; Jury M., et al. 435/7.1; 424/9.8 435/11 435/25 435/7.9 435/7.91 436/71 436/827. G01N033/535 G01N033/566 G01N033/92 .

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☐	L1	(botulinum same ($pigment or pigmentation)).clm.	1
☐	L2	(botulinum same ($color or color$)).clm.	6
☐	L3	L2 not l1	5
☐	L4	l1	1
☐	L5	(botulinum near10 ($pigment or pigmentation))	8
☐	L6	L5 not l1	7
☐	L7	((skin or hair) near5 pigmentation) or melasma or (skin near damage) or vitiligo or moles or dermatofibromas or (dermoid near cyst) or freckles or keloids or keratoacanthomas or lipomasmoles or nevi or (atypical near2 moles) or (dysplastic near nevi) or (pyogenic near granulomas) or (seborrheic near keratoses) (actinic near keratosis) or (skin near tags) or (pigment near disorders) or albinism, or (pigment near2 loss)	471856
☐	L8	botx or bont or botox or botulinum or botulin or bontoxilysin or botoxilysin-a or dysport or myobloc or btx or btxa or btx-a or bn or (clostridial near2 neurotoxin)	119436
☐	L9	L8 same l7	1076
☐	L10	L8 near50 l7	220
☐	L11	L8 near25 l7	182
☐	L12	L8 near15 l7	151
☐	L13	L8 near5 l7	88
☐	L14	L8 near10 l7	134
☐	L15	((((method or process).clm. same (skin or dermal or topically or topical or intradermal or melann or pigment or dermatofibromas or fibromas or dermoid or freckles or keloids or seborrheic or keratoses or actinic or albinism or melasma or vitiligo or damage).clm. and (botulin or botulinum or botox or bont or bonta or bnt or bonta or bnta or bontoxylysin or bottox or bot-tox or dysport or myobloc or neurotoxin).ti,ab,clm.) not ((method or process).clm. same (skin or dermal or topically or topical or intradermal or melann or pigment or dermatofibromas or fibromas or dermoid or freckles or keloids or seborrheic or keratoses or actinic or albinism or melasma or vitiligo or damage).clm. and (botulin or botulinum or botox or bont or bonta or bnt or bonta or bnta or bontoxylysin or bottox or bot-tox or dysport or myobloc or neurotoxin).ti,ab,clm. and allergan.asn. and (skin or dermal or topically or topical or intradermal or melann or pigment or dermatofibromas or fibromas or dermoid or freckles or keloids or seborrheic or keratoses or actinic or albinism or melasma or vitiligo or damage).clm. and (method or process).clm.)))	114

☐	L16	((((method or process).clm. same (skin or dermal or topically or topical or intradermal or melann or pigment or dermatofibromas or fibromas or dermoid or freckles or keloids or seborrheic or keratoses or actinic or albinism or melasma or vitiligo or damage).clm. and (botulin or botulinum or botox or bont or bonta or bnt or bonta or bnta or bontoxylysin or bottox or bot-tox or dysport or myobloc or neurotoxin).ti,ab,clm.) not ((method or process).clm. same (skin or dermal or topically or topical or intradermal or melann or pigment or dermatofibromas or fibromas or dermoid or freckles or keloids or seborrheic or keratoses or actinic or albinism or melasma or vitiligo or damage).clm. and (botulin or botulinum or botox or bont or bonta or bnt or bonta or bnta or bontoxylysin or bottox or bot-tox or dysport or myobloc or neurotoxin).ti,ab,clm. and allergan.asn. and (skin or dermal or topically or topical or intradermal or melann or pigment or dermatofibromas or fibromas or dermoid or freckles or keloids or seborrheic or keratoses or actinic or albinism or melasma or vitiligo or damage).clm. and (method or process).clm.)))	114
☐	L17	((((method or process).clm. same (skin or dermal or topically or topical or intradermal or melann or pigment or dermatofibromas or fibromas or dermoid or freckles or keloids or seborrheic or keratoses or actinic or albinism or melasma or vitiligo or damage).clm. and (botulin or botulinum or botox or bont or bonta or bnt or bonta or bnta or bontoxylysin or bottox or bot-tox or dysport or myobloc or neurotoxin).ti,ab,clm.) not ((method or process).clm. same (skin or dermal or topically or topical or intradermal or melann or pigment or dermatofibromas or fibromas or dermoid or freckles or keloids or seborrheic or keratoses or actinic or albinism or melasma or vitiligo or damage).clm. and (botulin or botulinum or botox or bont or bonta or bnt or bonta or bnta or bontoxylysin or bottox or bot-tox or dysport or myobloc or neurotoxin).ti,ab,clm. and allergan.asn. and (skin or dermal or topically or topical or intradermal or melann or pigment or dermatofibromas or fibromas or dermoid or freckles or keloids or seborrheic or keratoses or actinic or albinism or melasma or vitiligo or damage).clm. and (method or process).clm.)))	114
☐	L18	((((method or process).clm. same (skin or dermal or topically or topical or intradermal or melann or pigment or dermatofibromas or fibromas or dermoid or freckles or keloids or seborrheic or keratoses or actinic or albinism or melasma or vitiligo or damage).clm. and (botulin or botulinum or botox or bont or bonta or bnt or bonta or bnta or bontoxylysin or bottox or bot-tox or dysport or myobloc or neurotoxin).ti,ab,clm.) not ((method or process).clm. same (skin or dermal or topically or topical or intradermal or melann or pigment or dermatofibromas or fibromas or dermoid or freckles or keloids or seborrheic or keratoses or actinic or albinism or melasma or vitiligo or damage).clm. and (botulin or botulinum or botox or bont or bonta or bnt or bonta or bnta or bontoxylysin or bottox or bot-tox or dysport or myobloc or neurotoxin).ti,ab,clm. and allergan.asn. and (skin or dermal or topically or topical or intradermal or melann or pigment or dermatofibromas or fibromas or dermoid or freckles or keloids or seborrheic or keratoses or actinic or albinism or melasma or vitiligo or damage).clm. and (method or process).clm.)))	114
☐	L19	l10 not l18	219
☐	L20	l11 not l18	181
☐	L21	l19 not moles	4
☐	L22	l9 not moles	77

☐	L23	L22 not l18	77
☐	L24	l23 and l9.clm.	0
☐	L25	l23 and l8.clm.	8
☐	L26	l9 and l8.clm.	73
☐	L27	L26 not l25 not l23	65

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? s botulin? (25n) (albino or hyperpigment? or pigment? or hair?)
           72005  BOTULIN?
           91831  ALBINO
           21364  HYPERPIGMENT?
          660570  PIGMENT?
          469672  HAIR?
      S1     188  BOTULIN? (25N) (ALBINO OR HYPERPIGMENT? OR PIGMENT? OR
                  HAIR?)
? s s1/2004:2006
>>>One or more prefixes are unsupported
>>>  or undefined in one or more files.
>>>Year ranges not supported in one or more files
             187  S1
        16935702  PY=2004 : PY=2006
      S2      58  S1/2004:2006
? s s1 not s2
             188  S1
              58  S2
      S3     130  S1 NOT S2
? rd
      S4      74  RD  (unique items)
? t s4/kwic/all
>>>KWIC option is not available in file(s): 399

 4/KWIC/1     (Item 1 from file: 155)
DIALOG(R)File 155:(c) format only 2006 Dialog. All rts. reserv.

  ... pA/pF;  n  = 11) and reduced the extent of inactivation of the Ca(2+)
currents.    ***Botulinum***  toxin, an inhibitor of syntaxin, accelerated the
inactivation   profile   of   Ca(2+)   currents   in    ***hair***    cells.
Immunocytochemical data also indicated that the Ca(2+) channels and
syntaxin are co-localized...


 4/KWIC/2     (Item 2 from file: 155)
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  ... area were carefully noted and all patients had pretreatment and
posttreatment photographs. The effect of  ***botulinum***  toxin injections
on the horizontal brow rhytides was recorded by measuring the distance from
the frontal hairline to the superior edge of the eyebrow in the
mid-pupillary line. Patients were followed...


 4/KWIC/3     (Item 3 from file: 155)
DIALOG(R)File 155:(c) format only 2006 Dialog. All rts. reserv.

  Descriptors: *Botulinum Toxin Type A--administration and dosage--AD
;  *Collagen--administration  and  dosage--AD; *Hair Removal--nursing
--NU;  *Laser  Surgery--nursing--NU; *Professional Autonomy; *Sclerotherapy
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--nursing--NU; *Surgery, Plastic--nursing...

4/KWIC/4 (Item 4 from file: 155)
DIALOG(R)File 155:(c) format only 2006 Dialog. All rts. reserv.

... some blue spots on their cheeks. In four patients whose sweating had extended beyond the hairline, remnants of gustatory sweating showed up. Overall, the affected area of gustatory sweating could be reduced by botulinum toxin A from an average of 31 cm2 before treatment to 4 cm2 after treatment...

4/KWIC/5 (Item 5 from file: 155)
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... the options available for treating photoaged skin in 2001. Various lasers (e.g., vascular lesion, ***pigmented*** lesion, ***hair*** removal, and resurfacing), botulinum A toxin, chemical peels, and various dermal and subcutaneous filler substances all are discussed.
; Biocompatible Materials; Botulinum Toxin Type A--therapeutic use --TU; Chemexfoliation; Face; Hair Removal--methods--MT; Humans; Rhytidoplasty--methods--MT; Skin Diseases--therapy--TH

4/KWIC/6 (Item 6 from file: 155)
DIALOG(R)File 155:(c) format only 2006 Dialog. All rts. reserv.

... focus on some interesting aspects: (1) the necessity for an exact anamnesis before treatment with botulinum toxin to ensure correct treatment; (2) the advantages of Minor's test in special situations, for example, when sweating occurs in regions of hairy skin, retroauricular, at the back of the auricle and in areas distant from the site...

4/KWIC/7 (Item 7 from file: 155)
DIALOG(R)File 155:(c) format only 2006 Dialog. All rts. reserv.

[Presynaptic effects of botulinum toxin type A on the neuronally evoked response of albino and pigmented rabbit iris sphincter and dilator muscles]

4/KWIC/8 (Item 8 from file: 155)
DIALOG(R)File 155:(c) format only 2006 Dialog. All rts. reserv.

Descriptors: *Botulinum Toxin Type A--adverse effects--AE; *Hypopigmentation--chemically induced--CI; *Neuromuscular Agents--adverse effects--AE; *Skin--drug effects--DE; *Skin Pigmentation --drug effects--DE

4/KWIC/9 (Item 9 from file: 155)
DIALOG(R)File 155:(c) format only 2006 Dialog. All rts. reserv.

... acetylcholinesterase staining properties, and average fiber diameter were determined 5 weeks after varying doses of botulinum A toxin were administered into ***albino*** rabbit longissimus dorsi muscle. The average fiber diameter within the muscle appeared to be a function of the dose of ***botulinum*** toxin injected. Fiber diameter variability correlated with the dose of botulinum toxin administered. Both fiber...

4/KWIC/10 (Item 10 from file: 155)
DIALOG(R)File 155:(c) format only 2006 Dialog. All rts. reserv.

Histochemical effects of botulinum B toxin were studied on fibers from longissimus dorsi muscle in Albino rabbits and compared to effects produced by ***botulinum*** A toxin. Acetylcholinesterase staining, muscle fiber size analysis, and ATPase staining indicated botulinum B toxin produced a denervation gradient and field similar to that produced by botulinum A...

4/KWIC/11 (Item 11 from file: 155)
DIALOG(R)File 155:(c) format only 2006 Dialog. All rts. reserv.

Botulinum A toxin was injected into the superior rectus muscles of ***albino*** rabbits. Length-tension curves of detached superior rectus muscles were continuously measured with a length...

4/KWIC/12 (Item 12 from file: 155)
DIALOG(R)File 155:(c) format only 2006 Dialog. All rts. reserv.

The pupillary effects of retrobulbar injection of botulinum toxin A (oculinum) in ***albino*** rats.

4/KWIC/13 (Item 13 from file: 155)
DIALOG(R)File 155:(c) format only 2006 Dialog. All rts. reserv.

... rumen fluid of cattle suffering from botulism. The results were already available after seven hours. ***Botulinum*** toxin likewise could be identified from ***hair*** of a suspicious carcass. The investigations confirm the high sensitivity of this method.

4/KWIC/14 (Item 14 from file: 155)
DIALOG(R)File 155:(c) format only 2006 Dialog. All rts. reserv.

Descriptors: *Botulinum Toxins; *Eye Proteins--metabolism--ME; *GTP-Binding Proteins--metabolism--ME; *Heterotrimeric GTP-Binding Proteins ; *Membrane Proteins--metabolism--ME; *Retinal Pigments--metabolism --ME; *Rhodopsin--metabolism--ME
Chemical Name: Botulinum Toxins; Eye Proteins; GNAT1 protein, human ; Membrane Proteins; Retinal Pigments; Virulence Factors, Bordetella; Adenosine Diphosphate Ribose; Rhodopsin; ADP Ribose Transferases; exoenzyme C3, Clostridium botulinum; Pertussis Toxin; GTP-Binding Proteins; Heterotrimeric GTP-Binding Proteins

4/KWIC/15 (Item 15 from file: 155)
DIALOG(R)File 155:(c) format only 2006 Dialog. All rts. reserv.

... and acid phosphatase were studied histochemically in sections of hypoglossal nuclei of normal adult male albino rats, and rats from 1 to 56 days after axotomy of the left hypoglossal nerve, or after injection of ***botulinum*** toxin into the left side of the tongue. Both axotomy and botulinum caused a diminution...

4/KWIC/16 (Item 1 from file: 5)
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...ABSTRACT: 2 pA/pF; n = 11) and reduced the extent of inactivation of the Ca2+ currents. ***Botulinum*** toxin, an inhibitor of syntaxin,

accelerated the inactivation profile of Ca2+ currents in hair cells. Immunocytochemical data also indicated that the Ca2+ channels and syntaxin are co-localized in...

4/KWIC/17 (Item 2 from file: 5)
DIALOG(R)File 5:(c) 2007 The Thomson Corporation. All rts. reserv.

Effects of botulinum A toxin on the neuronally evoked response of albino and pigmented rabbit iris sphincter and dilator muscles

4/KWIC/18 (Item 3 from file: 5)
DIALOG(R)File 5:(c) 2007 The Thomson Corporation. All rts. reserv.

...ABSTRACT: rumen fluid of cattle suffering from botulism. The results were already available after seven hours. ***Botulinum*** toxin likewise could be identified from ***hair*** of a suspicious carcass. The investigations confirm the high sensitivity of this method.
DESCRIPTORS: CLOSTRIDIUM-BOTULINUM TOXIN TYPE C HAIR RUMEN FLUID

4/KWIC/19 (Item 4 from file: 5)
DIALOG(R)File 5:(c) 2007 The Thomson Corporation. All rts. reserv.

...ABSTRACT: Two processing conditions (short and extended heating) were also compared for their ability to enhance pigment color and eliminate the natural meat microbial population. Meat slurries varying in cure composition were inoculated with a composite of six different strains of Clostridium ***botulinum*** , types A and B. After processing, the packages were incubated at 10 and 27°..
DESCRIPTORS: CLOSTRIDIUM-BOTULINUM MODEL MEAT SYSTEMS FOOD PROCESSING SPORE OUTGROWTH TOXIN PRODUCTION PIGMENT COLOR MEAT FLORA

4/KWIC/20 (Item 5 from file: 5)
DIALOG(R)File 5:(c) 2007 The Thomson Corporation. All rts. reserv.

DESCRIPTORS: BOOK ANIMALS MAN CLOSTRIDIUM-BOTULINUM MICROORGANISMS BACTERIA COLOR CURING NITROSAMINES PIGMENTS STORAGE FLAVOR ASCORBATE TOXICOLOGY CARCINOGENS

4/KWIC/21 (Item 1 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

...Identifiers--DORSAL-ROOT GANGLIA; TOXIN TYPE-A; PALMAR-HYPERHIDROSIS; SKIN-RESPONSE; BOTULINUM TOXIN; HAIRLESS SKIN; INJURY; ORGANIZATION; DENERVATION; TRANSECTION

4/KWIC/22 (Item 2 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

...Abstract: and glycosaminoglycans, eliminate solar elastosis, and normalize epidermal atypia. Complications are largely related to infection, pigmentary alteration, delayed healing, and scarring, but appropriate patient screening and prophylaxis can minimize their incidence.

Botulinum toxin injections are used in dermatology for two main purposes: elimination or attenuation of dynamic...

4/KWIC/23 (Item 3 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

...Abstract: some blue spots on their cheeks. In four patients whose sweating had extended beyond the hairline, remnants of gustatory sweating showed up. Overall,the affected area of gustatory sweating could be reduced by botulinum toxin A from an average of 31 cm(2) before treatment to 4 cm(2...

4/KWIC/24 (Item 4 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

...Identifiers--CURED-MEAT; SODIUM-NITRITE; DINITROSYL FERROHEMOCHROME; MUSCLE FOODS; IRON; BOTULINUM; OXIDE; COMPLEXES; PIGMENT; FLAVOR

4/KWIC/25 (Item 5 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

...Identifiers--CARBON-DIOXIDE LASER; SOFT-TISSUE AUGMENTATION; BOTULINUM-A EXOTOXIN; HAIR TRANSPLANTATION; INJECTABLE COLLAGEN; SKIN; PROPHYLAXIS; INFECTION; IMPLANT; UPDATE

4/KWIC/26 (Item 6 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

...Identifiers--ANTERIOR ***HAIRLINE*** INCISION; ***BOTULINUM*** TOXIN; CORRUGATOR SUPERCILII; UPPER BLEPHAROPLASTY; MUSCLE RESECTION; EYEBROW PTOSIS; LIFT; RHYTIDOPLASTY; ANATOMY; RHYTIDECTOMY

4/KWIC/27 (Item 7 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

...Research Fronts: ARTIFICIAL CHROMOSOMES; ALZHEIMERS-DISEASE LOCUS AD3; DOWN-SYNDROME REGION; HUMAN GENOME PROJECT)
95-1373 001 (BOTULINUM TOXIN; RECIPROCAL INHIBITION; HAND MUSCLE REFLEXES FOLLOWING AIR-PUFF STIMULATION)
95-4957 001 (LINKAGE DISEQUILIBRIUM; MAPPING COMPLEX DISEASE TRAITS; CARTILAGE HAIR HYPOPLASIA GENE; CONGENITAL NEPHROTIC SYNDROME LOCUS; FINNISH POPULATION)

4/KWIC/28 (Item 8 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

Research Fronts: 95-2389 001 (MOLECULAR MECHANISMS IN SYNAPTIC VESICLE RECYCLING; NEUROTRANSMITTER RELEASE; BOTULINUM NEUROTOXIN POISONING; NEURONAL EXOCYTOTIC FUSION MACHINE)
95-5684 001 (RAT VESTIBULAR EPITHELIUM; CRISTAE AMPULLARES; CYTOSKELETAL PROTEINS IN HUMAN HAIR-CELLS)

4/KWIC/29 (Item 9 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

...Identifiers--NATURALLY-OCCURRING PERIODONTITIS; ***PIGMENTED*** BACTEROIDES; CLOSTRIDIUM-BOTULINUM; CATS; HYBRIDIZATION; PROPOSAL; ACID; DOGS

4/KWIC/30 (Item 10 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

Research Fronts: 92-1334 002 (ISOLATED OUTER HAIR-CELLS OF THE GUINEA-PIG COCHLEA; EVOKED OTOACOUSTIC EMISSIONS; HORSESHOE BATS ORGAN; MECHANOELECTRICAL TRANSDUCTION)
92-3373 002 (BOTULINUM TOXIN; AUDITORY BRAIN-STEM RESPONSE; SPASMODIC TORTICOLLIS)
92-7696 002 (MIDDLE-EAR TRANSMISSION; COCHLEA IN...

4/KWIC/31 (Item 11 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

Research Fronts: 92-3373 003 (BOTULINUM TOXIN; AUDITORY BRAIN-STEM RESPONSE; SPASMODIC TORTICOLLIS)
92-1334 002 (ISOLATED OUTER HAIR-CELLS OF THE GUINEA-PIG COCHLEA; EVOKED OTOACOUSTIC EMISSIONS; HORSESHOE BATS ORGAN; MECHANOELECTRICAL TRANSDUCTION)
92...

4/KWIC/32 (Item 12 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

...Research Fronts: BEHAVIORAL STATES; VIBROACOUSTIC STIMULATION; TERM FETUS; LOW-RISK PRETERM INFANTS)
92-1334 001 (ISOLATED OUTER HAIR-CELLS OF THE GUINEA-PIG COCHLEA; EVOKED OTOACOUSTIC EMISSIONS; HORSESHOE BATS ORGAN; MECHANOELECTRICAL TRANSDUCTION)
92-3373 001 (BOTULINUM TOXIN; AUDITORY BRAIN-STEM RESPONSE; SPASMODIC TORTICOLLIS)

4/KWIC/33 (Item 13 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

Research Fronts: 92-1334 002 (ISOLATED OUTER HAIR-CELLS OF THE GUINEA-PIG COCHLEA; EVOKED OTOACOUSTIC EMISSIONS; HORSESHOE BATS ORGAN; MECHANOELECTRICAL TRANSDUCTION)
92-3373 001 (BOTULINUM TOXIN; AUDITORY BRAIN-STEM RESPONSE; SPASMODIC TORTICOLLIS)

4/KWIC/34 (Item 14 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

Research Fronts: 92-1334 002 (ISOLATED OUTER HAIR-CELLS OF THE GUINEA-PIG COCHLEA; EVOKED OTOACOUSTIC EMISSIONS; HORSESHOE BATS ORGAN; MECHANOELECTRICAL TRANSDUCTION)
92-3373 002 (BOTULINUM TOXIN; AUDITORY BRAIN-STEM RESPONSE; SPASMODIC TORTICOLLIS)

4/KWIC/35 (Item 15 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

Research Fronts: 92-1334 001 (ISOLATED OUTER HAIR-CELLS OF THE GUINEA-PIG COCHLEA; EVOKED OTOACOUSTIC EMISSIONS; HORSESHOE BATS ORGAN; MECHANOELECTRICAL TRANSDUCTION)
92-3373 001 (BOTULINUM TOXIN; AUDITORY BRAIN-STEM RESPONSE; SPASMODIC TORTICOLLIS)

4/KWIC/36 (Item 16 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

...Research Fronts: TOXICITY)
90-0549 001 (DEPRESSION IN PARKINSONS-DISEASE; BASAL GANGLIA; MEMORY DEFICITS)
90-2373 001 (BOTULINUM TOXIN; PERSISTENT TARDIVE-DYSKINESIA; SPASMODIC TORTICOLLIS; ACUTE DRUG-INDUCED DYSTONIA; ABNORMAL INVOLUNTARY MOVEMENTS)
90-6099 001 (PIGMENTED DOPAMINERGIC-NEURONS IN PARKINSONS-DISEASE; OXIDATION OF BIOLOGICALLY-ACTIVE POLYHYDROXYPHENOLS (SUBSTITUTED CATECHOLS))
90-7543 001...

4/KWIC/37 (Item 17 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

Research Fronts: 89-1301 001 (ISOLATED COCHLEAR OUTER HAIR-CELLS; SPONTANEOUS OTOACOUSTIC EMISSIONS; ACOUSTIC STIMULATION OF THE CONTRALATERAL EAR)
89-4267 001 (ACTIN INVOLVEMENT; SMOOTH-MUSCLE CELLS; BOTULINUM-C2 TOXIN; PLANT CYTOSKELETON; INTERMEDIATE FILAMENTS; PLASMODIUM-FALCIPARUM INVITRO; EARLY EVENTS)
89-7006 001 (ACTIN...

4/KWIC/38 (Item 18 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

Research Fronts: 89-1469 001 (BOTULINUM TOXIN; FOCAL DYSTONIA; DELAYED VISUAL MATURATION)
89-6599 001 (SPECTRAL SENSITIVITY; DISCRIMINATION OF COLOR; CHROMATIC VISION; PSYCHOPHYSICAL DATA; HUE SIGNALS; MACULAR PIGMENT; ADDITIVITY TEST)

4/KWIC/39 (Item 19 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

Research Fronts: 89-6599 003 (SPECTRAL SENSITIVITY; DISCRIMINATION OF COLOR; CHROMATIC VISION; PSYCHOPHYSICAL DATA; HUE SIGNALS; MACULAR PIGMENT; ADDITIVITY TEST)
89-1469 001 (BOTULINUM TOXIN; FOCAL DYSTONIA; DELAYED VISUAL MATURATION)

4/KWIC/40 (Item 20 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

Research Fronts: 89-1469 001 (BOTULINUM TOXIN; FOCAL DYSTONIA; DELAYED VISUAL MATURATION)
89-6599 001 (SPECTRAL SENSITIVITY; DISCRIMINATION OF COLOR; CHROMATIC VISION; PSYCHOPHYSICAL DATA; HUE SIGNALS; MACULAR PIGMENT; ADDITIVITY TEST)

4/KWIC/41 (Item 21 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

Research Fronts: 89-1469 001 (BOTULINUM TOXIN; FOCAL DYSTONIA; DELAYED VISUAL MATURATION)

89-6599 001 (SPECTRAL SENSITIVITY; DISCRIMINATION OF COLOR; CHROMATIC VISION; PSYCHOPHYSICAL DATA; HUE SIGNALS; MACULAR PIGMENT; ADDITIVITY TEST)

4/KWIC/42 (Item 22 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

Research Fronts: 89-1469 001 (BOTULINUM TOXIN; FOCAL DYSTONIA; DELAYED VISUAL MATURATION)
89-6599 001 (SPECTRAL SENSITIVITY; DISCRIMINATION OF COLOR; CHROMATIC VISION; PSYCHOPHYSICAL DATA; HUE SIGNALS; MACULAR PIGMENT; ADDITIVITY TEST)

4/KWIC/43 (Item 23 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

Research Fronts: 89-2043 002 (FROG RETINA; HORIZONTAL CELLS; TIGER SALAMANDER CONE PHOTORECEPTORS)
89-1469 001 (BOTULINUM TOXIN; FOCAL DYSTONIA; DELAYED VISUAL MATURATION)
89-6599 001 (SPECTRAL SENSITIVITY; DISCRIMINATION OF COLOR; CHROMATIC VISION; PSYCHOPHYSICAL DATA; HUE SIGNALS; MACULAR PIGMENT; ADDITIVITY TEST)

4/KWIC/44 (Item 24 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

Research Fronts: 89-1469 002 (BOTULINUM TOXIN; FOCAL DYSTONIA; DELAYED VISUAL MATURATION)
89-6599 001 (SPECTRAL SENSITIVITY; DISCRIMINATION OF COLOR; CHROMATIC VISION; PSYCHOPHYSICAL DATA; HUE SIGNALS; MACULAR PIGMENT; ADDITIVITY TEST)

4/KWIC/45 (Item 25 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

Research Fronts: 89-6599 002 (SPECTRAL SENSITIVITY; DISCRIMINATION OF COLOR; CHROMATIC VISION; PSYCHOPHYSICAL DATA; HUE SIGNALS; MACULAR PIGMENT; ADDITIVITY TEST)
89-1469 001 (BOTULINUM TOXIN; FOCAL DYSTONIA; DELAYED VISUAL MATURATION)

4/KWIC/46 (Item 26 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

Research Fronts: 89-1095 002 (DUCHENNE MUSCULAR-DYSTROPHY; MOLECULAR DELETION PATTERNS; DMD GENE)
89-1469 001 (BOTULINUM TOXIN; FOCAL DYSTONIA; DELAYED VISUAL MATURATION)
89-6599 001 (SPECTRAL SENSITIVITY; DISCRIMINATION OF COLOR; CHROMATIC VISION; PSYCHOPHYSICAL DATA; HUE SIGNALS; MACULAR PIGMENT; ADDITIVITY TEST)

4/KWIC/47 (Item 27 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

Research Fronts: 88-1331 002 (OUTER HAIR-CELLS; GUINEA-PIG COCHLEA; BIDIRECTIONAL TRANSDUCTION CYCLE)

88-0915 001 (TARDIVE-DYSKINESIA IN BIPOLAR AFFECTIVE-DISORDER; BOTULINUM TOXIN; CRANIAL DYSTONIA; FACIAL SPASM; COCHLEAR NERVE)
88-4491 001 (COCHLEAR MODEL FOR ACOUSTIC EMISSIONS...

4/KWIC/48 (Item 28 from file: 34)
DIALOG(R)File 34:(c) 2007 The Thomson Corp. All rts. reserv.

Research Fronts: 88-0516 002 (DUCHENNE MUSCULAR-DYSTROPHY; X-LINKED RETINITIS PIGMENTOSA; GENE LOCUS)
88-0915 002 (TARDIVE-DYSKINESIA IN BIPOLAR AFFECTIVE-DISORDER; BOTULINUM TOXIN; CRANIAL DYSTONIA; FACIAL SPASM; COCHLEAR NERVE)
88-0134 001 (RETINOBLASTOMA GENE; WILMS TUMOR; RESTRICTION...

4/KWIC/49 (Item 1 from file: 45)
DIALOG(R)File 45:(c) 2007 Elsevier B.V. All rts. reserv.

DESCRIPTORS:
...silicone gel; thioctic acid; ubiquinone; unclassified drug; 2 hydroxyacid; alpha tocopherol; antioxidant; ascorbic acid; hydroxyacid; botulinum toxin A; botulinum toxin B; collagen implant; collagen; patient; hydrogel; hyperpigmentation; scar formation; keloid; liposuction; physical attractiveness; priority journal; preoperative evaluation; rejuvenation; scar; skin graft; skin...

4/KWIC/50 (Item 2 from file: 45)
DIALOG(R)File 45:(c) 2007 Elsevier B.V. All rts. reserv.

DESCRIPTORS:
cosmetic; retinoic acid; minoxidil; sibutramine; tetrahydrolipstatin; finasteride; botulinum toxin A; eflornithine; drug industry; hair growth; hypertension; lotion; medical ethics; priority journal; rejuvenation; prostate tumor; risk benefit analysis; short survey...

4/KWIC/51 (Item 3 from file: 45)
DIALOG(R)File 45:(c) 2007 Elsevier B.V. All rts. reserv.

DESCRIPTORS:
botulinum toxin A; cosmetic; finasteride; drug industry; medical decision making; hair growth; human; medical ethics; prescription; priority journal; rejuvenation; skin; health care organization; hair follicle; alopecia; aging; drug manufacture; plant; food and drug administration; forehead

4/KWIC/52 (Item 4 from file: 45)
DIALOG(R)File 45:(c) 2007 Elsevier B.V. All rts. reserv.

DESCRIPTORS:
...topical agent; avobenzone; anthranilic acid; 4 aminobenzoic acid; antioxidant; ascorbic acid; glycolic acid; benzophenone derivative; botulinum toxin A; cinnamic acid derivative; collagen; dibenzoylmethane derivative; skin cancer; sun exposure; skin abrasion; leisure; pigment disorder; phototherapy; skin defect; skin protection ; low level laser therapy; lentigo; skin disease; skin surgery...

4/KWIC/53 (Item 5 from file: 45)
DIALOG(R)File 45:(c) 2007 Elsevier B.V. All rts. reserv.

DESCRIPTORS:

...unclassified drug; valaciclovir; xipamide; water; antibiotic agent; adrenalin; aciclovir; antivirus agent; ascorbic acid; bleaching agent; botulinum toxin A; famciclovir; glycolic acid; YAG laser; treatment contraindication; tissue; morbidity; surgeon; fibrosis; patient; scar; pigment disorder; postoperative care; postoperative complication; laser surgery; infection; human; skin color; patient selection; practice guideline...

4/KWIC/54 (Item 6 from file: 45)
DIALOG(R)File 45:(c) 2007 Elsevier B.V. All rts. reserv.

DESCRIPTORS:
...clarithromycin; doxycycline; metronidazole; politef; tetracycline; 2 hydroxyacid; azelaic acid; benzoyl peroxide; emollient agent; antioxidant; antiandrogen; botulinum toxin; isotretinoin; retinoid; sunscreen; skin cancer; photoaging; skin care; hormonal therapy; patient; risk; soft tissue; wrinkle; hyperpigmentation; hospital patient; surgery; skin abrasion; humidifier; conference paper; treatment planning; sun exposure; primary prevention; blepharitis...

4/KWIC/55 (Item 7 from file: 45)
DIALOG(R)File 45:(c) 2007 Elsevier B.V. All rts. reserv.

DESCRIPTORS:
Lorenzo oil; histrelin; mercaptamine; nafarelin acetate; pegademase; retinoic acid; succimer; cladribine; carnitine; baclofen; botulinum antiserum; drug legislation; finance; hairy cell leukemia; food and drug administration; lead poisoning; precocious puberty; priority journal; spasticity; cystinosis; law...

4/KWIC/56 (Item 8 from file: 45)
DIALOG(R)File 45:(c) 2007 Elsevier B.V. All rts. reserv.

DESCRIPTORS:
pentamidine; nucleoside analog; zidovudine; dornase alfa; botulinum toxin; cladribine; priority journal; Pneumocystis pneumonia; Human immunodeficiency virus infection; hairy cell leukemia; drug cost; drug development; cystic fibrosis; blepharospasm; drug legislation

4/KWIC/57 (Item 1 from file: 71)
DIALOG(R)File 71:(c) 2007 Elsevier B.V. All rts. reserv.

...2 pA/pF; n = 11) and reduced the extent of inactivation of the CaSUP2+ currents. ***Botulinum*** toxin, an inhibitor of syntaxin, accelerated the inactivation profile of CaSUP2+ currents in ***hair*** cells. Immunocytochemical data also indicated that the CaSUP2+ channels and syntaxin are co-localized in...

4/KWIC/58 (Item 1 from file: 73)
DIALOG(R)File 73:(c) 2007 Elsevier B.V. All rts. reserv.

MEDICAL DESCRIPTORS:
*auditory nervous system; *hair cell; *Clostridium botulinum

4/KWIC/59 (Item 2 from file: 73)
DIALOG(R)File 73:(c) 2007 Elsevier B.V. All rts. reserv.

...was confirmed. These patients were treated with an intracutaneous

injection of 2.5 IU of ***botulinum*** toxin A (Botox, Allergan, USA) into the affected skin area. It was necessary to reinject the medicine into a small temporal hair-covered area in one patient, in whom Minor's test did not appear clearly positive...

4/KWIC/60 (Item 1 from file: 94)
DIALOG(R)File 94:(c)2006 Japan Science and Tech Corp(JST). All rts. reserv.

...ABSTRACT: and carbon dioxide laser treatment for spots, CP using glycolic acid and injection of the botulinus toxin for wrinkle, and monochloroacetate treatment for wart are described. Hyperhidrosis, ingrowing nails, ***hair*** growth, depilation, senile wart, etc. are described.

4/KWIC/61 (Item 2 from file: 94)
DIALOG(R)File 94:(c)2006 Japan Science and Tech Corp(JST). All rts. reserv.

...ABSTRACT: for prevention of senile change is discussed. Chemical peeling (CP), collagen injection, laser depilation and botulinus toxin injection are discussed. Effectiveness of laser treatment of pigmentary lesion and vascular lesion on the improvement in QOL of persons with dermatosis is emphasized...

4/KWIC/62 (Item 1 from file: 135)
DIALOG(R)File 135:(c) 2007 NewsRx. All rts. reserv.

...TEXT: 000); nose reshaping (177,000); and facelift (117,000). * The top 5 nonsurgical procedures were: ***botulinum*** toxin injection-BTX (1.6 million, up 2356% since 1997); chemical peel (1.4 million); collagen injection (1.1 million); microdermabrasion (915,000); and laser ***hair*** removal (855,000). * Women had over 7.4 million cosmetic procedures, 88% of the total...

4/KWIC/63 (Item 1 from file: 149)
DIALOG(R)File 149:(c) 2007 The Gale Group. All rts. reserv.

... 11,810

Note: Table made from bar graph.

Top 5 Cosmetic Nonsurgical Procedures in 2001

Botulinum Toxin Injection (Botox, Allergan; Myobloc, Elan Pharmaceuticals)	1,600,300
Chemical Peel	1,361,479
Collagen Injection	1,098,519
Microdermabrasion	915,312
Laser Hair Removal	854,582

Note: Table made from bar graph.

Top 5 Cosmetic Surgeries For Women...

4/KWIC/64 (Item 2 from file: 149)
DIALOG(R)File 149:(c) 2007 The Gale Group. All rts. reserv.

... c. Can correct deep wrinkles and skin laxity.
d. Effects persist for several years.
10. ***Botulinum*** toxin (BoTox):
a. Produces irregular ***pigmentation*** .
b. Is used as an adjunctive therapy with lasers.
c. Produces reversible paralysis of small...

4/KWIC/65 (Item 3 from file: 149)
DIALOG(R)File 149:(c) 2007 The Gale Group. All rts. reserv.

... safe) track to approval, and even some small FDA grants. Examples of approved orphans include botulinum toxin for blepharospasm, dornase alfa for cystic fibrosis, and cladribine for hairy cell leukaemia.
Japan is a welcome newcomer to the orphan drugs field, passing legislation two...

4/KWIC/66 (Item 1 from file: 156)
DIALOG(R)File 156:(c) format only 2006 Dialog. All rts. reserv.

Identifiers: cell adhesion; cell growth regulation; microinjection; visual photoreceptor; retinal pigment epithelium; cytokine; botulinum toxin; phosphorylation; protein tyrosine kinase; guanine nucleotide binding protein; fibronectin; integrin; cell senescence; cell line...

4/KWIC/67 (Item 1 from file: 266)
DIALOG(R)File 266:Comp & dist by NTIS, Intl Copyright All Rights Res. All rts. reserv.

DESCRIPTORS: tamoxifen; chick embryo; chicken; intracellular transport; calcium flux; chemical kinetics; polymerase chain reaction; cochlea; ear hair cell; electrophysiology; estrogen analog; gene expression; genetic regulatory element; immunocytochemistry; botulinum toxin; protein kinase A; protein structure function; protein transport; potassium channel; transfection /expression vector; calcium...

4/KWIC/70 (Item 1 from file: 434)
DIALOG(R)File 434:(c) 2006 The Thomson Corp. All rts. reserv.

Research Fronts: 86-4059 001 (LIPID-PEROXIDATION PRODUCTS; FREE-RADICAL OXIDATION; METABOLISM OF MALONDIALDEHYDE; FLUORESCENT PIGMENTS; LENS IN CATARACT; FORMATION OF ADDUCTS)
86-6417 001 (LIPID OXIDATION IN COOKED MEATS; CLOSTRIDIUM-BOTULINUM TOXIN PRODUCTION IN TURKEY FRANKFURTERS; REDUCED WATER ACTIVITY; STABILITY OF SORBIC ACID)

4/KWIC/71 (Item 2 from file: 434)
DIALOG(R)File 434:(c) 2006 The Thomson Corp. All rts. reserv.

...Research Fronts: LEUKOTRIENE GENERATION)
86-4059 001 (LIPID-PEROXIDATION PRODUCTS; FREE-RADICAL OXIDATION; METABOLISM OF MALONDIALDEHYDE; FLUORESCENT PIGMENTS; LENS IN CATARACT; FORMATION OF ADDUCTS)
86-6417 001 (LIPID OXIDATION IN COOKED MEATS; CLOSTRIDIUM-

BOTULINUM TOXIN PRODUCTION IN TURKEY FRANKFURTERS; REDUCED WATER ACTIVITY; STABILITY OF SORBIC ACID)

4/KWIC/72 (Item 3 from file: 434)
DIALOG(R)File 434:(c) 2006 The Thomson Corp. All rts. reserv.

...Research Fronts: INFANTS ACUITY; DIABETIC MACULAR EDEMA; RETINAL VISUAL-ACUITY; EPIKERATOPHAKIA IN CHILDREN; INFANT VISUAL PREFERENCES; RETINAL-PIGMENT EPITHELIUM)
86-5717 001 (BOTULINUM TOXIN; ESSENTIAL BLEPHAROSPASM; OCULAR MUSCLE DISORDERS; FACIAL SPASM)

4/KWIC/73 (Item 4 from file: 434)
DIALOG(R)File 434:(c) 2006 The Thomson Corp. All rts. reserv.

Research Fronts: 86-6417 002 (LIPID OXIDATION IN COOKED MEATS; CLOSTRIDIUM-BOTULINUM TOXIN PRODUCTION IN TURKEY FRANKFURTERS; REDUCED WATER ACTIVITY; STABILITY OF SORBIC ACID)
86-4059 001 (LIPID-PEROXIDATION PRODUCTS; FREE-RADICAL OXIDATION; METABOLISM OF MALONDIALDEHYDE; FLUORESCENT PIGMENTS; LENS IN CATARACT; FORMATION OF ADDUCTS)
86-4564 001 (METHYL LINOLENATE; LIPID OXIDATION-PRODUCTS; BUTYLATED...

4/KWIC/74 (Item 1 from file: 444)
DIALOG(R)File 444:(c) 2007 Mass. Med. Soc. All rts. reserv.

TEXT
...and are now used widely. (Ref. 2,3) The excellent response of axillary hyperhidrosis to botulinum toxin injections is due to the fact that the hyperactive sweat glands are usually localized in one or two small areas within the ***hair*** -bearing axillary skin. Since the toxin diffuses, causing a dose-dependent anhidrotic circle, two to...
?
PLEASE ENTER A COMMAND OR BE LOGGED OFF IN 5 MINUTES
? t s4/9/1 3 5 7 8 14 19 38 39 49 50 53 54 57 58 61 74

4/9/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2006 Dialog. All rts. reserv.

14171133 PMID: 12574487
Functional interaction of auxiliary subunits and synaptic proteins with Ca(v)1.3 may impart hair cell Ca2+ current properties.
Song Haitao; Nie Liping; Rodriguez-Contreras Adrian; Sheng Zu-Hang; Yamoah Ebenezer N
Center for Neuroscience, Department of Otolaryngology, University of California, Davis, California 95616, USA.
Journal of neurophysiology (United States) Feb 2003, 89 (2) p1143-9, ISSN 0022-3077--Print Journal Code: 0375404
Contract/Grant No.: DC-03828; DC; NIDCD
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS; Toxbib
We assessed the functional determinants of the properties of L-type Ca(2+) currents in hair cells by co-expressing the pore-forming Ca(V)1.3alpha(1) subunit with the auxiliary subunits beta(1A) and/or alpha(2delta). Because Ca(2+) channels in hair cells are poised to interact with synaptic proteins, we also co-expressed the Ca(V)1.3alpha(1) subunit

with syntaxin, vesicle-associated membrane protein (VAMP), and synaptosome associated protein of 25 kDa (SNAP25). Expression of the Ca(V)1.3alpha(1) subunit in human embryonic kidney cells (HEK 293) produced a dihydropyridine (DHP)-sensitive Ca(2+) current (peak current density -2.0 +/- 0.2 pA/pF; n = 11). Co-expression with beta(1A) and alpha(2delta) subunits enhanced the magnitude of the current (peak current density: Ca(V)1.3alpha(1) + beta(1A) = -4.3 +/- 0.8 pA/pF, n = 10; Ca(V)1.3alpha(1) + beta(1A) + alpha(2delta) = -4.1 +/- 0.6 pA/pF, n = 9) and produced a leftward shift of approximately 9 mV in the voltage-dependent activation of the currents. Furthermore, co-expression of Ca(V)1.3alpha(1) with syntaxin/VAMP/SNAP resulted in at least a twofold increase in the peak current density (-4.7 +/- 0.2 pA/pF; n = 11) and reduced the extent of inactivation of the Ca(2+) currents. ***Botulinum*** toxin, an inhibitor of syntaxin, accelerated the inactivation profile of Ca(2+) currents in ***hair*** cells. Immunocytochemical data also indicated that the Ca(2+) channels and syntaxin are co-localized in hair cells, suggesting there is functional interaction of the Ca(V)1.3alpha(1) with auxiliary subunits and synaptic proteins, that may contribute to the distinct properties of the DHP-sensitive channels in hair cells.

Descriptors: *Calcium--metabolism--ME; *Calcium Channels, L-Type --metabolism--ME; *Hair Cells--physiology--PH; *Synapses--physiology--PH; Animals; Botulinum Toxins--pharmacology--PD; Calcium Channel Blockers --pharmacology--PD; Calcium Channels; Calcium Channels, L-Type--chemistry --CH; Calcium Channels, L-Type--genetics--GE; Cell Line; Dihydropyridines --pharmacology--PD; Humans; Ion Channel Gating--physiology--PH; Kidney --cytology--CY; Membrane Potentials--drug effects--DE; Membrane Potentials --physiology--PH; Membrane Proteins--antagonists and inhibitors--AI; Membrane Proteins--metabolism--ME; Nerve Tissue Proteins--metabolism--ME; Qa-SNARE Proteins; R-SNARE Proteins; Rana catesbeiana; Recombinant Proteins --chemistry--CH; Recombinant Proteins--genetics--GE; Recombinant Proteins --metabolism--ME; Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.; Synaptosomal-Associated Protein 25; Transfection

CAS Registry No.: 0 (Botulinum Toxins); 0 (Calcium Channel Blockers); 0 (Calcium Channels); 0 (Calcium Channels, L-Type); 0 (Dihydropyridines); 0 (Membrane Proteins); 0 (Nerve Tissue Proteins); 0 (Qa-SNARE Proteins); 0 (R-SNARE Proteins); 0 (Recombinant Proteins) ; 0 (SNAP25 protein, human); 0 (Synaptosomal-Associated Protein 25); 0 (botulinum toxin type C); 166872-16-8 (Cacna1d protein, rat); 3337-17-5 (1,4-dihydropyridine); 7440-70-2 (Calcium)

Record Date Created: 20030207

Record Date Completed: 20030404

4/9/3 (Item 3 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

13564662 PMID: 11995305

Laser hair removal, sclerotherapy, injection of collagen and botox.

Florida nurse (United States) Sep 2001, 49 (3) p30, ISSN 0015-4199 --Print Journal Code: 16930510R

Publishing Model Print

Document type: News

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: NURSING; Toxbib

Descriptors: *Botulinum Toxin Type A--administration and dosage--AD ; *Collagen--administration and dosage--AD; *Hair Removal--nursing --NU; *Laser Surgery--nursing--NU; *Professional Autonomy; *Sclerotherapy --nursing--NU; *Surgery, Plastic--nursing--NU; Florida; Humans; Laser Surgery--legislation and jurisprudence--LJ; Licensure, Nursing--legislation and jurisprudence--LJ; Surgery, Plastic--legislation and jurisprudence--LJ

CAS Registry No.: 0 (Botulinum Toxin Type A); 9007-34-5 (Collagen)

Record Date Created: 20020508
Record Date Completed: 20020610

4/9/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13371972 PMID: 11535427
Lasers and cosmetic dermatologic surgery for aging skin.
Rohrer T E
Section of Dermatologic Surgery, Department of Dermatology, Boston University School of Medicine, Boston, Massachusetts 02118, USA.
Clinics in geriatric medicine (United States) Nov 2001, 17 (4) p769-94, vii, ISSN 0749-0690--Print Journal Code: 8603766
Publishing Model Print
Document type: Journal Article; Review
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS; Toxbib
Many topical agents and physical modalities have been used throughout the years to give the face a more youthful appearance. The goal has always been to effectively and consistently rejuvenate the face while minimizing the time of recovery and risk for complications. Because each person is unique, there is no one modality that is best for everyone. This article reviews some of the options available for treating photoaged skin in 2001. Various lasers (e.g., vascular lesion, ***pigmented*** lesion, ***hair*** removal, and resurfacing), botulinum A toxin, chemical peels, and various dermal and subcutaneous filler substances all are discussed. (86 Refs.)
Descriptors: *Laser Surgery; *Skin--surgery--SU; *Skin Aging--pathology --PA; *Skin Diseases--surgery--SU; Biocompatible Materials; Botulinum Toxin Type A--therapeutic use--TU; Chemexfoliation; Face; Hair Removal--methods--MT; Humans; Rhytidoplasty--methods--MT; Skin Diseases --therapy--TH
CAS Registry No.: 0 (Biocompatible Materials); 0 (Botulinum Toxin Type A)
Record Date Created: 20010905
Record Date Completed: 20011218

4/9/7 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13198655 PMID: 11329944
[Presynaptic effects of botulinum toxin type A on the neuronally evoked response of albino and pigmented rabbit iris sphincter and dilator muscles]
Ishikawa H; Mitsui Y; Yoshitomi T; Mashimo K; Aoki S; Mukuno K; Shimizu K
Department of Ophthalmology Kitasato University, School of Medicine, 1-15-1 Kitasato, Sagamihara, 228-8555, Japan.
Nippon Ganka Gakkai zasshi (Japan) Apr 2001, 105 (4) p218-22, ISSN 0029-0203--Print Journal Code: 7505716
Publishing Model Print
Document type: Journal Article ; English Abstract
Languages: JAPANESE
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS; Toxbib
PURPOSE: To investigate the effects of botulinum toxin type A(botulinum A toxin) on the autonomic and other non-adrenergic, non-cholinergic nerve terminals. METHODS: The effects of neurotoxin on twitch contractions evoked by electrical field stimulation (EFS) were studied in isolated rabbit iris

sphincter and dilator muscles using isometric tension recording. RESULTS: Botulinum A toxin(150 nM) inhibited the fast cholinergic and slow substance P-ergic component of contraction evoked by EFS in the rabbit iris sphincter muscle without affecting the response to carbachol and substance P. Botulinum A toxin(150 nM) did not affect the twitch contraction evoked by EFS in the rabbit iris dilator muscle. CONCLUSION: These data indicated that botulinum A toxin may inhibit not only the acetylcholine release in the cholinergic nerve terminals, but also substance P release from the trigeminal nerve terminals of the rabbit iris sphincter muscle. However, neurotoxin has little effect on the adrenergic nerve terminals of the rabbit iris dilator muscle.

Tags: Male

Descriptors: *Botulinum Toxin Type A--pharmacology--PD; *Iris --drug effects--DE; *Presynaptic Terminals--drug effects--DE; Animals; English Abstract; In Vitro; Muscles--drug effects--DE; Rabbits

CAS Registry No.: 0 (Botulinum Toxin Type A)

Record Date Created: 20010501

Record Date Completed: 20010719

4/9/8 (Item 8 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

12341705 PMID: 10098540

Prevalence of periocular depigmentation after repeated botulinum toxin A injections in African American patients.

Roehm P C; Perry J D; Girkin C A; Miller N R

Neuro-Ophthalmology Unit, The Johns Hopkins Medical Institutions, Baltimore, Maryland, USA.

Journal of neuro-ophthalmology - the official journal of the North American Neuro-Ophthalmology Society (UNITED STATES) Mar 1999, 19 (1) p7-9, ISSN 1070-8022--Print Journal Code: 9431308

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS; Toxbib

Botulinum toxin A (Botox), administered by subcutaneous or intramuscular injection, is the most commonly used and most successful medication for many craniocervical dystonias. Although some patients experience side effects related to the neuroparalytic action of the medication, these side effects are temporary. In 1996, permanent periocular cutaneous depigmentation was reported in three white patients after repeated Botox injections, suggesting that loss or alteration of melanin pigment might be a permanent side effect of long-term Botox injections. The authors examined and photographed 26 African American patients who were receiving periocular Botox injections for hemifacial spasm and essential blepharospasm. The authors found no evidence of periocular cutaneous depigmentation in any of these patients.

Descriptors: *Botulinum Toxin Type A--adverse effects--AE; *Hypopigmentation--chemically induced--CI; *Neuromuscular Agents--adverse effects--AE; *Skin--drug effects--DE; *Skin Pigmentation --drug effects--DE; Adult; African Continental Ancestry Group; Aged; Aged, 80 and over; Blepharospasm--drug therapy--DT; Botulinum Toxin Type A--therapeutic use--TU; Hemifacial Spasm--drug therapy--DT; Humans; Hypopigmentation --ethnology--EH; Injections; Maryland--epidemiology--EP; Middle Aged; Neuromuscular Agents--therapeutic use--TU; Prevalence; Skin--pathology--PA

CAS Registry No.: 0 (Botulinum Toxin Type A); 0 (Neuromuscular Agents)

Record Date Created: 19990423

Record Date Completed: 19990423

4/9/14 (Item 14 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08444363 PMID: 2110532
Interaction of small G proteins with photoexcited rhodopsin.
Wieland T; Ulibarri I; Aktories K; Gierschik P; Jakobs K H
Pharmakologisches Institut der Universitat Heidelberg, FRG.
FEBS letters (NETHERLANDS) Apr 24 1990, 263 (2) p195-8, ISSN 0014-5793--Print Journal Code: 0155157
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS; Toxbib
Bovine rod outer segment (ROS) membranes contain in addition to the heterotrimeric G protein transducin, several small GTP-binding proteins (23-27 kDa). Furthermore, these membranes contain two substrate proteins (about 22 and 24 kDa) for botulinum C3 ADP-ribosyltransferase known to ADP-ribosylate small G proteins in any mammalian cell type studied so far. Most interestingly, [32P]ADP-ribosylation of ROS membrane small G proteins by C3 is regulated by light and guanine nucleotides in a manner similar to pertussis toxin-catalyzed [32P]ADP-ribosylation of the alpha-subunit of transducin. These findings suggest that not only the heterotrimeric G protein transducin but also the C3 substrate small G proteins present in ROS membranes interact with photoexcited rhodopsin and thus contribute to its signalling action.
Descriptors: *Botulinum Toxins; *Eye Proteins--metabolism--ME; *GTP-Binding Proteins--metabolism--ME; *Heterotrimeric GTP-Binding Proteins ; *Membrane Proteins--metabolism--ME; *Retinal Pigments--metabolism --ME; *Rhodopsin--metabolism--ME; ADP Ribose Transferases--metabolism--ME; Adenosine Diphosphate Ribose--metabolism--ME; Animals; Cattle; Electrophoresis, Polyacrylamide Gel; Light; Molecular Weight; Pertussis Toxin; Research Support, Non-U.S. Gov't; Retina--metabolism--ME; Virulence Factors, Bordetella--metabolism--ME
CAS Registry No.: 0 (Botulinum Toxins); 0 (Eye Proteins); 0 (GNAT1 protein, human); 0 (Membrane Proteins); 0 (Retinal Pigments); 0 (Virulence Factors, Bordetella); 20762-30-5 (Adenosine Diphosphate Ribose); 9009-81-8 (Rhodopsin)
Enzyme No.: EC 2.4.2.- (ADP Ribose Transferases); EC 2.4.2.- (exoenzyme C3, Clostridium botulinum); EC 2.4.2.31 (Pertussis Toxin); EC 3.6.1.- (GTP-Binding Proteins); EC 3.6.1.46 (Heterotrimeric GTP-Binding Proteins)
Record Date Created: 19900611
Record Date Completed: 19900611

4/9/19 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0005264610 BIOSIS NO.: 198682110997
AN EVALUATION OF ANTIBOTULINAL ACTIVITY IN NITRITE-FREE CURING SYSTEMS CONTAINING DINITROSYL FERROHEMOCHROME
AUTHOR: WOOD D S (Reprint); COLLINS-THOMPSON D L; USBORNE W R; PICARD B
AUTHOR ADDRESS: DEP ENVIRON BIOL, UNIV GUELPH, GUELPH, ONT, CAN N1G 2W1** CANADA
JOURNAL: Journal of Food Protection 49 (9): p691-695 1986
ISSN: 0362-028X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: The ability of the pigment dinitrosyl ferrohemochrome to mimic the cured meat color function attributed to nitrite, was evaluated in a number of nitrite-free, model meat systems. In addition, compounds with reported antibotulinal properties were compared to the antibotulinal effect of nitrite. Fifteen treatments were evaluated and compared to 50 and 150 ppm nitrite. Two processing conditions (short and extended heating) were also compared for their ability to enhance pigment color and eliminate the natural meat microbial population. Meat slurries varying in cure composition were inoculated with a composite of six different strains of Clostridium ***botulinum*** , types A and B. After processing, the packages were incubated at 10 and 27° C, and were analyzed for toxin. The treatment containing 3000 ppm sodium hypophosphite most closely resembled the 150 ppm nitrite control in its ability to prevent spore outgrowth and toxin production. The treatment containing 1250 ppm monomethyl fumerate also scored better than the other treatments including ethylene diamine tetraacetic acid (EDTA), potassium sorbate and tertiary butyl hydroquinome (TBHQ), but was slightly less inhibitory than sodium hypophosphite. The longer heat treatment eliminated all the natural meat flora (lactic acid bacteria) and enhanced the color production of the pigment.

REGISTRY NUMBERS: 14797-65-0: NITRITE; 26202-48-2: DINITROSYL FERROHEMOCHROME
DESCRIPTORS: CLOSTRIDIUM-BOTULINUM MODEL MEAT SYSTEMS FOOD PROCESSING SPORE OUTGROWTH TOXIN PRODUCTION PIGMENT COLOR MEAT FLORA
DESCRIPTORS:
MAJOR CONCEPTS: Foods; Infection; Toxicology
BIOSYSTEMATIC NAMES: Endospore-forming Gram-Positives--Eubacteria, Bacteria, Microorganisms
COMMON TAXONOMIC TERMS: Bacteria; Eubacteria; Microorganisms
CHEMICALS & BIOCHEMICALS: NITRITE; DINITROSYL FERROHEMOCHROME
CONCEPT CODES:
10010 Comparative biochemistry
10050 Biochemistry methods - General
10060 Biochemistry studies - General
10064 Biochemistry studies - Proteins, peptides and amino acids
10066 Biochemistry studies - Lipids
10069 Biochemistry studies - Minerals
10614 External effects - Temperature as a primary variable
13012 Metabolism - Proteins, peptides and amino acids
13516 Food technology - Meats and meat by-products
13530 Food technology - Evaluations of physical and chemical properties
13532 Food technology - Preparation, processing and storage
17501 Muscle - General and methods
22502 Toxicology - Foods, food residues, additives and preservatives
23001 Temperature - General measurement and methods
30500 Morphology and cytology of bacteria
31000 Physiology and biochemistry of bacteria
32000 Microbiological apparatus, methods and media
36001 Medical and clinical microbiology - General and methods
36002 Medical and clinical microbiology - Bacteriology
37006 Public health - Public health laboratory methods
37060 Public health: disease vectors - Inanimate
37400 Public health: microbiology - Public health microbiology
39002 Food microbiology - Food and beverage spoilage and contamination
39500 Disinfection, disinfectants and sterilization
BIOSYSTEMATIC CODES:
07810 Endospore-forming Gram-Positives

01327193 Genuine Article#: GP751 Number of References: 46
Title: THE INVARIANCE OF UNIQUE WHITE - A POSSIBLE IMPLICATION FOR NORMALIZING CONE ACTION SPECTRA
Author(s): WALRAVEN J; WERNER JS
Corporate Source: TNO,INST PERCEPT,KAMPWEG 5/3769 DE SOESTERBERG//NETHERLANDS/; UNIV COLORADO,DEPT PSYCHOL/BOULDER//CO/80309
Journal: VISION RESEARCH, 1991, V31, N12, P2185-2193
Language: ENGLISH Document Type: ARTICLE
Geographic Location: NETHERLANDS; USA
Subfile: SciSearch; CC LIFE--Current Contents, Life Sciences
Journal Subject Category: NEUROSCIENCES; OPHTHALMOLOGY
Abstract: The locus of unique white was measured for two observers over a wide range of stimulus intensities, using both direct matching and absolute judgements. Chromaticity coordinates of each observer's unique white were found to be invariant over a range of about 4 log units. This result is discussed in the context of the class of models that explain the intensity-evoked change in perceived color (Bezold-Brucke effect) on the basis of response compression in color channels. Invariance of unique white may be interpreted, then, as evidence for its non-polarizing effect on color channels, including the three classes of cones. On the basis of that assumption we have used unique white as criterion for normalizing cone action spectra. Assuming an equal-energy spectrum for the standard observer's unique-white stimulus, as we found to be consistent with literature data, we calculated the associated coefficients of the conversion formulae between CIE space and cone space (both for XYZ to LMS and LMS to XYZ) of the Vos-Walraven (1971 Vision Research, 11, 799-818) and Smith-Pokorny (1975 Vision Research, 15, 161-171) receptor fundamentals. The action spectra that thus result, intersect at the spectral neutral (achromatic) points of the three classes of dichromates.
Identifiers--KeyWords Plus: HUE CANCELLATION; COLOR; SENSITIVITY; ADDITIVITY; MODELS
Research Fronts: 89-1469 001 (BOTULINUM TOXIN; FOCAL DYSTONIA; DELAYED VISUAL MATURATION)
89-6599 001 (SPECTRAL SENSITIVITY; DISCRIMINATION OF COLOR; CHROMATIC VISION; PSYCHOPHYSICAL DATA; HUE SIGNALS; MACULAR PIGMENT; ADDITIVITY TEST)
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4/9/39 (Item 19 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

01290056 Genuine Article#: GL732 Number of References: 103
Title: EFFECTS OF CHROMATIC ADAPTATION ON OPPONENT INTERACTIONS IN MONKEY INCREMENT-THRESHOLD SPECTRAL-SENSITIVITY FUNCTIONS
Author(s): KALLONIATIS M; HARWERTH RS
Corporate Source: UNIV HOUSTON,COLL OPTOMETRY/HOUSTON//TX/77204
Journal: JOURNAL OF THE OPTICAL SOCIETY OF AMERICA A-OPTICS AND IMAGE SCIENCE, 1991, V8, N11, P1818-1831
Language: ENGLISH Document Type: REVIEW
Geographic Location: USA
Subfile: SciSearch; CC PHYS--Current Contents, Physical, Chemical & Earth Sciences; CC ENGI--Current Contents, Engineering, Technology & Applied Sciences
Journal Subject Category: OPTICS
Abstract: The effects of chromatic adaptation on the opponent interactions of cone mechanisms were investigated by using increment-threshold spectral-sensitivity (ITSS) functions and threshold-versus-radiance (TVR) curves in rhesus monkey subjects. The TVR curves showed shape- and field-sensitivity invariance for both 580- and 500-nm adapting backgrounds and indicated that three cone mechanisms were mediating detection over moderate adapting-field intensity levels. Differential adaptation between the long-wavelength-sensitive (L) and the middle-wavelength-sensitive (M) opponent (L - M) and nonopponent (L + M) channels and the short-wavelength-sensitive (S) channel caused changes in the shape of the ITSS function as the adapting-field intensity was increased without changes in the level of cone interaction. Chromatic adaptation also resulted in significant changes in the shape of the ITSS functions, but it still exhibited characteristic L-M opponent interactions. Converting ITSS data to cone-contrast coordinates for R-G adapting fields indicated that the relative contribution of the L and M cones at the second site was approximately equal (detection contour slope almost-equal-to 1). Consequently, most of the changes in the shape of ITSS functions under chromatic adaptation are explained by the von Kries adaptation principle. ITSS functions on a green background also exhibited opponent interactions between S cones and longer-wavelength cones. The cone-contrast coordinates, when expressed for S cones, showed that the inhibitory interactions occur because the S-cone signal subtracts from

both M and L cones.
Identifiers--KeyWords Plus: RED-GREEN; PROCESS ADDITIVITY; PI-MECHANISMS; RHESUS-MONKEY; LUMINANCE FLICKER; LIGHT ADAPTATION; FIELD ADDITIVITY; COLOR MECHANISMS; VISUAL DETECTION; GANGLION-CELLS
Research Fronts: 89-6599 003 (SPECTRAL SENSITIVITY; DISCRIMINATION OF COLOR; CHROMATIC VISION; PSYCHOPHYSICAL DATA; HUE SIGNALS; MACULAR PIGMENT; ADDITIVITY TEST)
89-1469 001 (BOTULINUM TOXIN; FOCAL DYSTONIA; DELAYED VISUAL MATURATION)
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4/9/49 (Item 1 from file: 45)
DIALOG(R)File 45:EMCare

01388894 EMCare No: 38008030
Cosmetic considerations and nonlaser cosmetic procedures in ethnic skin
Jackson B.A.
Dr. B.A. Jackson, Skin Wellness Center of Chicago, 111 N. Wabash Ave, Chicago, IL 60602 United States
Dermatologic Clinics (DERMATOL. CLIN.) (United States) 2003, 21/4 (703-712)
CODEN: DRMCD ISSN: 0733-8635
DOCUMENT TYPE: Journal ; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 72
RECORD TYPE: Abstract

The face of the aesthetic patient is changing to be more representative of the ethnic diversity of the United States population. It is imperative that the cosmetic dermatologic surgeon not only understand the concerns of the ethnic aesthetic patient but have an awareness of the unique needs of those with darker skin.

BRAND NAME/MANUFACTURER NAME: mederma/Merz/France; botox/Allergan/United States; myobloc/Elan/United States
MANUFACTURER NAMES: Merz/France; Allergan/United States; Elan/United States
DEVICE BRAND NAME/MANUFACTURER NAME: Avogel/Avocet Polymer Technologies/United States; Zyderm/Collagen Corporation/United States; Zyplast/Collagen Corporation/United States; Artecoll/Rofil/United States; Hyalaform Gel/Biomatrix/United States; Restylane/Q Med/Sweden; Nokor/Becton Dickinson/United States; Steristrips/3M/United States; Dermabond/Ethicon/United States; Dermologen/Collagenesis/United States; Alloderm/Lifecell/United States; Cymetra/Lifecell/United States; Facian/Medical Aesthetics/United States
DEVICE MANUFACTURER NAMES: Avocet Polymer Technologies/United States; Collagen Corporation/United States; Rofil/United States; Biomatrix/United States; Q Med/Sweden; Becton Dickinson/United States; 3M/United States; Ethicon/United States; Collagenesis/United States; Lifecell/United States; Medical Aesthetics/United States
DESCRIPTORS:
*cosmetic; *skin
hyaluronic acid derivative; onion extract; plant extract; poly(methyl methacrylate); retinol; silicone gel; thioctic acid; ubiquinone; unclassified drug; 2 hydroxyacid; alpha tocopherol; antioxidant; ascorbic acid; hydroxyacid; botulinum toxin A; botulinum toxin B; collagen implant; collagen; patient; hydrogel; hyperpigmentation; scar formation; keloid; liposuction; physical attractiveness; priority journal; preoperative evaluation; rejuvenation; scar; skin graft; skin surgery; ethnic group; United States; population; surgeon; acne; adjuvant therapy; aging; body image; hypertrophic scar; implant; chloasma; cultural factor; human

4/9/50 (Item 2 from file: 45)
DIALOG(R)File 45:EMCare

01328705 EMCare No: 37064280
In the eye of the beholder
Pearson H.
Nature (NATURE) (United Kingdom) 28 AUG 2003, 424/6952 (990-991)
CODEN: NATUA ISSN: 0028-0836
DOCUMENT TYPE: Journal ; Short Survey
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
RECORD TYPE: Abstract

Does the pharmaceutical industry's future lie at the boundary between drugs and cosmetics? Or is the prospect of effective 'cosmeceuticals' a beauty myth? Helen Pearson investigates.

BRAND NAME/MANUFACTURER NAME: propecia/Merck Sharp and Dohme; xenical/Hoffmann La Roche; meridia/Abbott; renova; vaniqa; botox; rogaine
MANUFACTURER NAMES: Merck Sharp and Dohme; Hoffmann La Roche; Abbott; Integriderm/United States; Galderma/Switzerland; Phytopharm/United Kingdom; Pfizer; Anaderm

DESCRIPTORS:
*eye
cosmetic; retinoic acid; minoxidil; sibutramine; tetrahydrolipstatin; finasteride; botulinum toxin A; eflornithine; drug industry; hair growth; hypertension; lotion; medical ethics; priority journal; rejuvenation; prostate tumor; risk benefit analysis; short survey; skin; United States; esthetics; literature; acne; African trypanosomiasis; aging; alopecia; drug approval; drug classification; drug cost; human; drug indication; plant; food and drug administration

4/9/53 (Item 5 from file: 45)
DIALOG(R)File 45:EMCare

00978223 EMCare No: 32924288
Erbium:YAG cutaneous laser resurfacing
Alster T.S.; Lupton J.R.
Dr. T.S. Alster, Washington Inst. Derm. Laser Surg., 2311 M Street NW, Washington, DC 20037 United States
Dermatologic Clinics (DERMATOL. CLIN.) (United States) 2001, 19/3 (453-466)
CODEN: DRMCD ISSN: 0733-8635
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 123
RECORD TYPE: Abstract

The short-pulsed Er:YAG laser system is an excellent ablative tool for cutaneous resurfacing. This system is most efficacious for patients with milder cutaneous involvement, including mild photoinduced facial rhytides, mildly atrophic scars, and textural changes caused by fibrosis and dermatochalasis. The Er:YAG laser cannot achieve the same dramatic clinical and histologic improvements produced with the COSUB2 laser but does offer some distinct advantages that make it a valuable addition to the laser surgeon's armamentarium. The Er:YAG laser, because of its higher affinity for water-containing tissues, effects a much finer level of tissue ablation. Although erbium laser resurfacing results in decreased postoperative morbidity with a shorter recovery period, it cannot effect the same degree of improvement in photodamaged skin as can the COSUB2 laser. Excellent results, however, can be achieved with this laser, up to 50% or more overall clinical improvement, in patients with milder photodamage and scarring (Glogau classes I and II). In darker-skinned patients, the Er:YAG laser is often the preferred treatment modality. Continued research in the field has already led to the development of longer-pulsed Er:YAG lasers, which offer a compromise between the COSUB2 laser and the short-pulsed Er:YAG lasers in terms of clinical benefits while maintaining the safety profile of the traditional short-pulsed system. In addition, many surgeons now use a combination approach with the COSUB2 and Er:YAG lasers in an effort to maximize collagen contraction in certain areas and limit postoperative morbidity. As more research is conducted within the field of cutaneous resurfacing, newer systems will be developed in the continuing effort to create the ideal laser system - one which ameliorates the signs of photoaging without risk of major side effects or significant postoperative recovery.

BRAND NAME/MANUFACTURER NAME: botox/Allergan/United States; aquaphor; catrix 10; elta; theraplix
MANUFACTURER NAMES: Allergan/United States
DEVICE BRAND NAME/MANUFACTURER NAME: Contour/Sciton/United States; Vigilon; Second Skin
DEVICE MANUFACTURER NAMES: Sciton/United States
DESCRIPTORS:

*erbium; *laser
collagen; petrolatum; macrogol; lidocaine; hexamidine isetionate; hydroquinone; retinoic acid; unclassified drug; valaciclovir; xipamide; water; antibiotic agent; adrenalin; aciclovir; antivirus agent; ascorbic acid; bleaching agent; botulinum toxin A; famciclovir; glycolic acid; YAG laser; treatment contraindication; tissue; morbidity; surgeon; fibrosis ; patient; scar; pigment disorder; postoperative care; postoperative complication; laser surgery; infection; human; skin color; patient selection; practice guideline; preoperative care; priority journal; rejuvenation; sweat gland disease; treatment indication; wound care; collagen synthesis; safety; scar formation; skin; hospital patient; photoaging; risk; side effect; acne; analgesia; erythema; edema; erbium YAG laser; esthetic surgery

4/9/54 (Item 6 from file: 45)
DIALOG(R)File 45:EMCare

00609775 EMCare No: 29228428
A lifetime of healthy skin: Implications for women
Bergfeld W.F.
Dr. W.F. Bergfeld, Department of Dermatology/Pathology, Cleveland Clinic Foundation, Cleveland, OH 44106 United States
International Journal of Fertility and Women's Medicine (INT. J. FERTIL. WOMEN'S MED.) (United States) 1999, 44/2 (83-95)
CODEN: IJWMF ISSN: 1069-3130
DOCUMENT TYPE: Journal ; Conference Paper
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 49
RECORD TYPE: Abstract

During her lifetime, a woman faces the possibility of seeking dermatological assistance for a myriad of conditions, including acne, rosacea, striae, photodamage, and skin cancers. It is important for clinicians and patients to be aware of the symptoms of these conditions as well as the most beneficial approaches for prevention, diagnosis, treatment, and management. The life expectancy of women has increased and predictions for the year 2050 estimate the average age at 81 years. This will place women at greater risk for dermatological problems, especially photodamage and skin cancer. In addition, various ethnic groups may manifest these conditions differently. Although acne is most prevalent among teenaged males, most can expect clearing by age 25. Females may continue to experience acne into the adult years, sometimes beyond the age of 40. Although it is not a life- threatening disease, acne may have psychosocial and quality-of-life consequences. Treatments for acne can be topical or systemic, and include retinoids, antibiotics, benzoyl peroxide, azelaic acid, and hormonal therapy. Rosacea is more common in women (especially during menopause) than in men. It is a chronic condition that can cause complications, including telangiectasia, conjunctivitis, and blepharitis. Although there is no cure, rosacea can be managed and controlled with medication. Topical antibiotics, such as metronidazole, and systemic antibiotics, such as tetracycline, clarithromycin, and doxycycline, are used to manage rosacea. Striae, or stretch marks, occur most frequently in pregnant women, adolescents experiencing growth spurts, weight lifters, and the obese. Although not a health threat, they can be psychologically distressing. There are not many treatment options for striae, but topical tretinoin and the pulsed dye laser offer promising results. Intrinsic, or normal, aging of the skin results from the process of chronological aging. Photodamage is skin damage caused by chronic exposure to ultraviolet (UV) light. It is the leading cause of extrinsic aging, or alterations of the skin due to environmental exposure. Estimates indicate that almost half of a person's UV exposure occurs by age 18. Photoaging causes numerous histologic, physiologic, and clinical changes;

it also increases the risk for skin cancer. Photodamage can be prevented through the use of sun screens, protective clothing, and avoidance of the sun during peak intensity time. The only product approved by the FDA for the treatment of photodamage (fine wrinkles, mottled hyperpigmentation, and skin roughness), topical tretinoin emollient cream, may help prevent additional photoaging when it is used to treat existing photoaging. Other management options for photodamaged skin include alpha-hydroxy acids, antioxidants, anti-androgens, moisturizers, and exfoliants. In patients with excessive manifestations of photodamage, surgical management may be needed, including dermabrasion, chemical peels, soft tissue augmentation, laser resurfacing, botulism toxin, and Gortex(R) threads. Clinicians must educate their patients about the most appropriate skin care regimen as well as approaches for preventing and treating common afflictions. In this way, women will have the best opportunity for having and maintaining healthy skin.

BRAND NAME/MANUFACTURER NAME: retin a
DESCRIPTORS:
*lifespan; *female; *rosacea; *stria; *acne; *skin; *aging
antibiotic agent; retinoic acid; clarithromycin; doxycycline; metronidazole ; politef; tetracycline; 2 hydroxyacid; azelaic acid; benzoyl peroxide; emollient agent; antioxidant; antiandrogen; botulinum toxin; isotretinoin; retinoid; sunscreen; skin cancer; photoaging; skin care; hormonal therapy; patient; risk; soft tissue; wrinkle; hyperpigmentation; hospital patient; surgery; skin abrasion; humidifier; conference paper; treatment planning; sun exposure; primary prevention; blepharitis; laser; antibiotic therapy; human; drug choice; laser surgery; patient counseling; patient education; photodermatosis; rhytidoplasty; food and drug administration; growth acceleration; adult; male; ethnic group; prediction; life expectancy; protective clothing; diagnosis; prevention; drug therapy; menopause; quality of life; telangiectasia; conjunctivitis; adolescent; pregnant woman; weight; health; pulsed dye laser; skin defect; long term exposure; ultraviolet radiation; environmental exposure; exposure; priority journal; skin protection

4/9/57 (Item 1 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

02263577 2003047716
Functional interaction of auxiliary subunits and synaptic proteins with CaSUBV1.3 May impart hair cell CaSUP2+ current properties
Song H.; Nie L.; Rodriguez-Contreras A.; Sheng Z.-H.; Yamoah E.N.
ADDRESS: E.N. Yamoah, Center for Neuroscience, Department of Otolaryngology, University of California, 1544 Newton Ct., Davis, CA 95616, United States
EMAIL: enyamoah@ucdavis.edu
Journal: Journal of Neurophysiology, 89/2 (1143-1149), 2003, United States
PUBLICATION DATE: February 1, 2003
CODEN: JONEA
ISSN: 0022-3077
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 43

We assessed the functional determinants of the properties of L-type CaSUP2+ currents in hair cells by co-expressing the pore-forming CaSUBv1.3alphaSUB1 subunit with the auxiliary subunits betaSUB1A and/or alphaSUB2delta. Because CaSUP2+ channels in hair cells are poised to interact with synaptic proteins, we also co-expressed the CaSUBv1.3alphaSUB1 subunit with syntaxin, vesicle-associated membrane protein (VAMP), and synaptosome associated protein of 25 kDa (SNAP25). Expression of the CaSUBv1.3alphaSUB1

subunit in human embryonic kidney cells (HEK 293) produced a dihydropyridine (DHP)-sensitive CaSUP2+ current (peak current density - 2.0 +/- 0.2 pA/pF; n = 11). Co-expression with betaSUB1A and alphaSUB2delta, subunits enhanced the magnitude of the current (peak current density: CaSUBv1.3alphaSUB1 + betaSUB1A = -4.3 +/- 0.8 pA/pF, n = 10; CaSUBv1.3alphaSUB1 + betaSUB1A + alphaSUB2delta = -4.1 +/- 0.6 pA/pF, n = 9) and produced a leftward shift of approximately 9 mV in the voltage-dependent activation of the currents. Furthermore, co-expression of CaSUBv1.3alphaSUB1 with syntaxin/ VAMP/SNAP resulted in at least a twofold increase in the peak current density (-4.7 +/- 0.2 pA/pF; n = 11) and reduced the extent of inactivation of the CaSUP2+ currents. ***Botulinum*** toxin, an inhibitor of syntaxin, accelerated the inactivation profile of CaSUP2+ currents in ***hair*** cells. Immunocytochemical data also indicated that the CaSUP2+ channels and syntaxin are co-localized in hair cells, suggesting there is functional interaction of the CaSUBv1.3alphaSUB1 with auxiliary subunits and synaptic proteins, that may contribute to the distinct properties of the DHP-sensitive channels in hair cells.

CLASSIFICATION CODE AND DESCRIPTION:
88.1.12.1 - NEUROSCIENCE / CELLULAR NEUROSCIENCE / Proteins / Structure
88.2.1.4 - NEUROSCIENCE / NERVE STRUCTURE AND FUNCTION / Physiology of Nerve Cells / Synaptic transmission
88.8.4.3 - NEUROSCIENCE / SENSORY SYSTEMS / Auditory System / Hair cell and transduction

4/9/58 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

11835016 EMBASE No: 2002407098
Rescue of auditory hair cells from aminoglycoside toxicity by Clostridium difficile toxin B, an inhibitor of the small GTPases Rho/Rac/Cdc42
Bodmer D.; Brors D.; Pak K.; Gloddek B.; Ryan A.F.
A.F. Ryan, Department of Surgery, UCSD School of Medicine, VA Medical Center, 9500 Gilman Drive #0666, San Diego, CA 92093 United States
AUTHOR EMAIL: afryan@ucsd.edu
Hearing Research (HEAR. RES.) (Netherlands) 2002, 172/1-2 (81-86)
CODEN: HERED ISSN: 0378-5955
PUBLISHER ITEM IDENTIFIER: S0378595502005142
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 36

The hair cells (HCs) are the most vulnerable elements in the cochlea and damage to them is the most common cause of sensorineural hearing loss. Understanding the intracellular events that lead to the death of HCs is a key to developing protective strategies. Recently, it has been shown that the c-Jun-N-terminal kinase (JNK) pathway is activated in HCs in response to aminoglycosides (J. Neurosci. 20 (2000) 43). We have studied the upstream events leading to JNK activation in aminoglycoside toxicity in vitro. The small GTPases Rac and Cdc42 are well known upstream activators of JNK in other cell types. Clostridium difficile toxin B monoglucosylates all members of the Rho GTPase subfamily (Rho, Rac and Cdc42 isoforms) and inhibits GTP binding by steric interference (Nature 341 (1989) 209). Organ of Corti explants from p5 rat basal turns were maintained in tissue culture and treated with C. difficile toxin B for 12 h. They were then treated with toxin B plus gentamicin for 72 h. Significantly less HC death was observed compared to with gentamicin alone. Toxin B alone had no effect on HCs at the highest concentration used. Using antibodies against phospho-c-Jun, we observed background immunoreactivity in control explants, strong staining of outer hair cell nuclei in gentamicin treated explants, and weaker immunostaining in explants treated with gentamicin and C. difficile toxin B. We conclude that Rho family small GTPases play a role in aminoglycoside

toxicity signaling as upstream activators of the JNK signaling pathway. (c) 2002 Elsevier Science B.V. All rights reserved.

DRUG DESCRIPTORS:
*bacterial toxin--drug combination--cb; *bacterial toxin--drug comparison --cm; *bacterial toxin--pharmacology--pd; *guanosine triphosphatase; *Rho factor; *Rac protein; *protein Cdc42
aminoglycoside; protein inhibitor; stress activated protein kinase; gentamicin--drug combination--cb; gentamicin--drug comparison--cm; gentamicin--pharmacology--pd; antibody; unclassified drug
MEDICAL DESCRIPTORS:
*auditory nervous system; *hair cell; *Clostridium botulinum toxicity; enzyme activation; in vitro study; Corti organ; stereospecificity ; cell type; binding affinity; inhibition kinetics; tissue culture; cell death; immunoreactivity; drug effect; staining; cell nucleus; immunohistochemistry; nonhuman; rat; controlled study; animal tissue; animal cell; article; priority journal
DRUG TERMS (UNCONTROLLED): toxin b--drug combination--cb; toxin b--drug comparison--cm; toxin b--pharmacology--pd
CAS REGISTRY NO.: 9059-32-9 (guanosine triphosphatase); 155215-87-5 (stress activated protein kinase); 1392-48-9, 1403-66-3, 1405-41-0 (gentamicin)
SECTION HEADINGS:
  004 Microbiology: Bacteriology, Mycology, Parasitology and Virology
  011 Otorhinolaryngology
  030 Clinical and Experimental Pharmacology
  037 Drug Literature Index

4/9/61 (Item 2 from file: 94)
DIALOG(R)File 94:JICST-EPlus
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05341668 JICST ACCESSION NUMBER: 02A0949775 FILE SEGMENT: JICST-E
Treatment in Dermatology for the Improvement of Patients' QOL. III. Practice in Private Clinics. 1. Treatment to Improve Patients' QOL in a Private Clinic.
UEDA SETSUKO (1)
(1) Uedasetsukokurinikku(fukuokashi)
Hifuka no Rinsho(Rinsho Derma (Tokyo), 2002, VOL.44,NO.11, PAGE.1357-1367, FIG.16, TBL.1, REF.12
JOURNAL NUMBER: Z0122BAV ISSN NO: 0018-1404
UNIVERSAL DECIMAL CLASSIFICATION: 616.5-08
LANGUAGE: Japanese COUNTRY OF PUBLICATION: Japan
DOCUMENT TYPE: Journal
ARTICLE TYPE: Commentary
MEDIA TYPE: Printed Publication
ABSTRACT: Treatment to improve patients' QOL in the beauty dermatology is described. In order to improve QOL of the healthy skin and maintain the youthful skin, the protection of ultraviolet ray is important. The patch test of cosmetics for prevention of senile change is discussed. Chemical peeling (CP), collagen injection, laser depilation and ***botulinus*** toxin injection are discussed. Effectiveness of laser treatment of pigmentary lesion and vascular lesion on the improvement in QOL of persons with dermatosis is emphasized. CP of acne and xanthoma is described. Finally, this paper indicates that fundamentals and experience of the dermatology are essential for the beauty dermatologist.
DESCRIPTORS: skin disease; qualty of life; cosmetic surgery; skin(animal tissue); ultraviolet radiation; dermatology; cosmetic; senile change; patch test; collagen; laser therapy; unhairing; botulinus toxin; nevus; keratosis; hemangioma; rosacea; acne; xanthoma; physician; integumentary preparation; human(primates); plastic surgery(technique); aliphatic carboxylic acid; aliphatic chlorine compound
IDENTIFIERS: practitioner; chemexfoliation

BROADER DESCRIPTORS: disease; life(livelihood); operative surgery; epithelial tissue; animal tissue; biomedical tissue; organization; light; electromagnetic wave; wave motion; radioactive ray; medicine; natural science; science; perfumery and cosmetics; aging(physiology); test; scleroprotein; animal protein; protein; therapy; laser application; utilization; removal; exotoxin; bacterial toxin; microorganism toxin; poison; toxic substance; matter; benign tumor; tumor; vascular disease; cardiovascular disease; vascular tissue tumor; metabolic disease; medical worker; job classified employee; worker; drug; carboxylic acid; aliphatic halogen compound; organohalogene compound; organochlorine compound
CLASSIFICATION CODE(S): GF05010P

4/9/74 (Item 1 from file: 444)
DIALOG(R)File 444:New England Journal of Med.

00122766

Botulinum Toxin, Sweating, and Body Odor (Correspondence)

Bushara, Magda Amin; Bushara, Khalaf; Heckmann, Marc; Plewig, Gerd; Pause, Bettina M.
The New England Journal of Medicine
Aug 22, 2002; 347 (8),pp 620-621
LINE COUNT: 00049 WORD COUNT: 00680
ISSN: 0028-4793

TEXT

Letter 001

To the Editor: Since our description of the anhidrotic effect of injections of botulinum toxin A in humans, (Reference 1) there have been several studies confirming the efficacy and safety of botulinum toxin injections in several forms of focal hyperhidrosis, including the study reported in the Journal by Heckmann et al. (Feb. 15, 2001, issue). (Reference 2) The injections have been shown to be particularly useful in axillary hyperhidrosis and are now used widely. (Reference 2,3) The excellent response of axillary hyperhidrosis to botulinum toxin injections is due to the fact that the hyperactive sweat glands are usually localized in one or two small areas within the ***hair*** -bearing axillary skin. Since the toxin diffuses, causing a dose-dependent anhidrotic circle, two to three injections are usually sufficient to denervate the oval hair-bearing area of the axilla without decreasing the efficacy of the toxin. (Reference 4) However, in most published studies, as many as 14 injections were used, which makes the procedure unnecessarily painful. We recommend the use of only two to four injections in each axilla in order to minimize discomfort and risk.|Magda Amin Bushara, M.D.Botulin ClinicEden Prairie, MN 55344|Khalaf Bushara, M.D.Minneapolis Veterans Affairs Medical CenterMinneapolis, MN 55417busha001@umn.edu

The authors reply:|

Letter 002

To the Editor: Bushara et al. deserve credit for showing that as few as two injections of botulinum toxin A per axilla suffice to produce anhidrosis in healthy volunteers. (Reference 1) The rate of axillary sweat production is typically below 25 mg per minute in normohidrotic persons but can reach 1000 mg per minute in patients with hyperhidrosis. In view of this difference, it seems appropriate to adhere to injection protocols that have proven efficacy for treating severe axillary hyperhidrosis. When 30-gauge needles were used for 10 injections, 98.6 percent of our patients rated their tolerance of treatment as excellent or good.

There is a second difference between normohidrotic persons and those

with hyperhidrosis: the former often report having unpleasant axillary odor, whereas the latter typically do not. (Reference 2) Body odor is attributed to the production by apocrine axillary sweat glands of a turbid secretion that has a pungent smell when it is degraded by microbes that are resident in the skin. (Reference 3) It has been suggested that apocrine activity in persons with hyperhidrosis does not parallel the activity of eccrine and apoeccrine glands. (Reference 2) Some patients with hyperhidrosis become more aware of their body odor after botulinum toxin injections. In contrast, a substantial reduction in body odor was found in normohidrotic healthy volunteers in whom one axilla was injected with botulinum toxin A and then compared with the other axilla. (Reference 4) This finding further supports the concept that normohidrotic persons and those with hyperhidrosis differ in the quantity and quality of sweat secretion. Both the extent of moisture and the extent of odor of the axillary milieu differ. Therefore, the effects of botulinum toxin injections on the two variables are not the same in healthy volunteers and patients with hyperhidrosis.|Marc Heckmann, M.D.Gerd Plewig, M.D.Ludwig Maximilians UniversityD-80337 Munich, Germanyheckmann@derma.med.uni-muenchen.de|Bettina M. Pause, Ph.D.Christian Albrecht University24098 Kiel, Germany

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Reference 005|
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?

neuroglia (noo-rog′lē-ă)

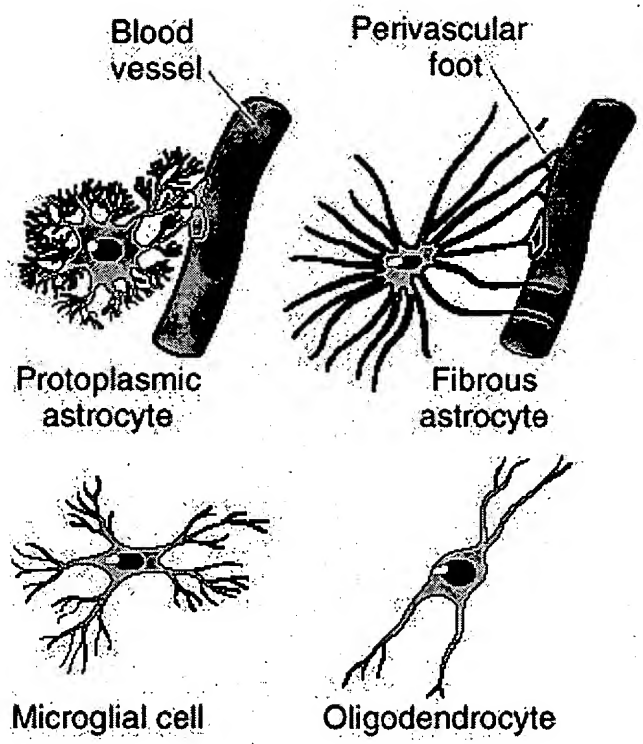
Non-neuronal cellular elements of the central and peripheral nervous system; formerly believed to be merely supporting cells but now thought to have important metabolic functions, since they are invariably interposed between neurons and the blood vessels supplying the nervous system. In central nervous tissue they include oligodendroglia cells, astrocytes, ependymal cells, and microglia cells. The satellite cells of ganglia and the neurolemmal or Schwann cells around peripheral nerve fibers can be interpreted as the oligodendroglia cells of the peripheral nervous system. Syn: glia, reticulum 2 *TA*, Kölliker reticulum

[neuro- + G. *glia,* glue]

Prev

neurogenous (neu·rog·e·nous) (nŏŏ -roj´ə-nəs) neurogenic.

neuroglia (neu·rog·lia) (nŏŏ -rog´le-ə) [*neuro-* + *-glia*] [TA] the supporting structure of nervous tissue. It consists of a fine web of tissue made up of modifi ectodermal elements, in which are enclosed peculiar branched cells known as *neuroglial cells* or *glial cells*. The neuroglial cells are of three types: astrocytes oligodendrocytes (astroglia and oligodendroglia), which appear to play a role in formation, transport of material to neurons, and maintenance of the ionic envirc of neurons; and microcytes (microglia), which phagocytize waste products of n tissue. Called also *glia*. See plate accompanying *nerve*.



■**Neuroglia, showing various types of neuroglial cells.**

interfascicular n. oligodendroglia of white matter along the myelin sheaths.

peripheral n. the neurilemma, Schwann cells, and satellite cells of the peripheral nervous system.

neuroglial (neu·rog·li·al) (nŏŏ -rog´le-əl) pertaining to the neuroglia.

neurogliocyte (neu·rog·lio·cyte) (nŏŏ -rog´le-o-sīt") [*neuroglia* + *-cyte*] a the neuroglia.

neurogliocytoma (neu·rog·lio·cy·to·ma) (nŏŏ -rog"le-o-si-to´mə) glioma

neuroglioma (neu·rog·li·o·ma) (nŏŏ -rog"le-o´mə) glioma.

Previous Doc Next Doc Go to Doc#
First Hit

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L6: Entry 39 of 114 File: PGPB Sep 8, 2005

DOCUMENT-IDENTIFIER: US 20050196414 A1
TITLE: Compositions and methods for topical application and transdermal delivery of botulinum toxins

Abstract Paragraph:
A composition for topical application of a botulinum toxin (including botulinum toxin derivatives) comprises a botulinum toxin and a carrier comprising a polymeric backbone comprising a long-chain polypeptide or nonpeptidyl polymer having attached positively charged branching or "efficiency" groups. The invention also relates to methods for reducing muscle paralysis and other conditions that may be treated with a botulinum toxin, particularly paralysis of subcutaneous, and most particularly, facial, muscles, by topically applying an effective amount of the botulinum toxin and carrier, in conjunction, to the subject's skin or epithelium. Kits for administration are also described.

CLAIMS:

1. A composition comprising a botulinum toxin and a carrier comprising a polymeric backbone having attached positively charged branching groups, wherein the association between the carrier and the botulinum toxin is non-covalent.

2. A composition according to claim 1 in which the botulinum toxin is a botulinum toxin derivative.

3. A composition according to claim 1 in which the botulinum toxin comprises a recombinant botulinum toxin.

4. A composition according to claim 1 in which the botulinum toxin comprises a modified botulinum toxin.

5. A composition according to claim 1 in which the botulinum toxin is selected from botulinum toxin serotypes A, B, C, D, E, F and G.

6. A composition according to claim 5 in which the botulinum toxin is botulinum toxin A.

7. A composition according to claim 5 in which the botulinum toxin is botulinum toxin B.

8. A composition according to claim 5 in which the botulinum toxin is botulinum toxin C.sub.1.

9. A composition according to claim 5 in which the botulinum toxin is botulinum toxin D.

10. A composition according to claim 5 in which the botulinum toxin is botulinum toxin E.

41. A kit for administration of a botulinum toxin to a subject comprising a

botulinum toxin and an effective amount for transdermal delivery thereof, of a carrier comprising a polymeric backbone having attached positively charged branching groups, wherein the association between the carrier and the botulinum toxin is non-covalent.

45. A kit according to claim 41 comprising a pre-formulated composition comprising the botulinum toxin and the carrier.

46. A kit according to claim 41 in which the botulinum toxin and the carrier are separately formulated for combining prior to administration.

47. A kit according to claim 41 in which the botulinum toxin is contained in a device for administering the botulinum toxin to a subject via the skin.

49. A kit for administration of a botulinum toxin to a subject comprising a device for delivering the botulinum toxin to the skin and a composition comprising a carrier comprising a polymeric backbone having attached positively charged branching groups selected from -(gly).sub.n1-(arg).sub- .n2, HIV-TAT and fragments thereof, and Antennapedia PTD, in which the subscript n1 is an integer of from 0 to about 20, and the subscript n2 is independently an odd integer of from about 5 to about 25.

51. A method of administering a botulinum toxin to a subject comprising topically applying to the skin or epithelium of the subject the botulinum toxin in conjunction with an effective amount of a carrier comprising a polymeric backbone having attached positively charged branching groups, wherein the association between the carrier and the botulinum toxin is non-covalent.

52. A method according to claim 51 comprising topically applying to the skin or epithelium of the subject an effective amount of a composition according to claim 1.

53. A method according to claim 51 in comprising separately applying the botulinum toxin and the carrier to the skin or epithelium of the subject.

54. A method according to claim 51 in which the botulinum toxin is administered to achieve a desired biologic effect.

55. A method according to claim 54 in which the botulinum toxin is administered to achieve an aesthetic or cosmetic benefit.

56. A method according to claim 54 in which the botulinum toxin is applied to reduce or prevent an immune response.

58. A method according to claim 54 in which the botulinum toxin is administered for prevention or reduction of symptoms associated with subjective or clinical hyperhidrosis.

59. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of subjective or clinical dystonic contractions or dystonia.

60. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with muscle spasm.

61. A method according to claim 60 in which the botulinum toxin is applied topically to the lower back of the subject.

62. A method according to claim 60 in which the botulinum toxin is topically applied to the neck of the subject.

63. A method according to claim 60 in which the botulinum toxin is topically applied to at least one leg of the subject.

64. A method according to claim 51 in which the botulinum toxin is applied topically to the face of the subject, or to a portion thereof.

65. A method according to claim 51 in which the botulinum toxin is applied topically to the axilla of the subject, or to a portion thereof.

66. A method according to claim 51 in which the botulinum toxin is applied topically to the palms of the hands or to the feet of the subject, or to a portion thereof.

67. A method according to claim 51 in which the botulinum toxin is applied topically to the back or neck of the subject, or to a portion thereof.

68. A method according to claim 51 in which the botulinum toxin is applied topically to the groin of the subject, or to a portion thereof.

69. A method according to claim 51 in which the composition is applied topically to the hands or feet of the subject, or to a portion thereof.

70. A method according to claim 51 in which the botulinum toxin is applied topically to the elbows, upper arms, knees, or upper legs of the subject, or to a portion thereof.

71. A method according to claim 51 in which the botulinum toxin is applied topically to the buttocks of the subject or to a portion thereof.

72. A method according to claim 51 in which the botulinum toxin is applied topically to the torso of the subject or to a portion thereof.

73. A method according to claim 51 in which the botulinum toxin is applied topically to the pelvis of the subject or to a portion thereof.

74. A method according to claim 51 in which the botulinum toxin is applied to generate or enhance an immune response.

75. A method according to claim 51 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with migraine headache.

76. A method according to claim 51 in which the botulinum toxin is applied topically for prevention or reduction of acne.

77. A method according to claim 51 in which the botulinum toxin is a botulinum toxin derivative.

78. A method according to claim 51 in which the botulinum toxin comprises a recombinant botulinum toxin.

79. A method according to claim 51 in which the botulinum toxin comprises a modified botulinum toxin.

80. A method according to claim 51 in which the botulinum toxin is selected from botulinum toxin serotypes A, B, C, D, E, F and G.

81. A method according to claim 51 in which the botulinum toxin is botulinum toxin A.

82. A method according to claim 51 in which the botulinum toxin is botulinum toxin B.

83. A method according to claim 51 in which the botulinum toxin is botulinum toxin C.

84. A method according to claim 51 in which the botulinum toxin is botulinum toxin D.

85. A method according to claim 51 in which the botulinum toxin is botulinum toxin E.

108. A method according to claim 51 in which the botulinum toxin comprises a recombinant botulinum toxin.

109. A method according to claim 51 in which the botulinum toxin is applied in a composition having a pH of from about 4.5 to about 6.3.

110. A method according to claim 51 in which the botulinum toxin is applied in a controlled release composition.

111. A method according to claim 51 in which the botulinum toxin is contained in a liquid composition.

112. A method according to claim 51 in which the botulinum toxin is contained in a gel composition.

113. A method according to claim 51 in which the botulinum toxin is contained in a composition that is a cream, lotion or ointment.

114. A method according to claim 51 in which the botulinum toxin is contained in a composition further comprising saline.

115. A method according to claim 51 in which the botulinum toxin is contained in a composition further comprising saline and a pH buffer system.

116. A method according to claim 51 in which the botulinum toxin is contained in a device for dispensing the botulinum toxin, which device is applied topically to the skin or epithelium of the subject.

117. A method according to claim 116 in which the device is a skin patch.

119. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with mucous secretion.

120. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of obesity or symptoms thereof.

121. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of inflammation or symptoms thereof.

122. A method according to claim 121 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with psoriasis.

124. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of snoring.

125. A method according to claim 54 in which the botulinum toxin is applied

topically for prevention or reduction of cutaneous symptoms associated with diabetes.

126. A method according to claim 54 in which the botulinum toxin is applied topically for improvement of wound healing.

127. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with autonomic nerve dysfunction.

128. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with cerebral palsy.

129. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with Hashimoto's thyroiditis.

130. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with mammary gland disorders.

131. A method according to claim 54 in which the botulinum toxin is applied topically for alteration of hair growth.

132. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with parathyroid disorders.

133. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with movement disorders.

134. A method according to claim 133 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with parkinson's disease.

135. A method according to claim 133 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with tremors.

136. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with epilepsy.

137. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with inner ear disorders.

138. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with urologic disorders.

139. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of other cholinergic-controlled secretions.

140. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with neuropshychiatric disorders.

141. A method according to claim 54 in which the botulinum toxin is applied topically for prevention or reduction of symptoms associated with injured muscles.

107 As noted above, abnormalities in a subject's autonomic nervous system include those characterized by an abnormally high parasympathetic activity or abnormally low parasympathetic activity and/or an abnormally high sympathetic activity or abnormally low sympathetic activity. There are numerous conditions that the inventors of the subject invention have, unexpectedly, discovered are at least partially manifested by abnormal balance of the sympathetic and parasympathetic functions of the autonomic nervous system, particularly those that manifest higher than normal (as defined by those seen in healthy individuals between the ages of about 20 to about 25 years old) ratio of sympathetic function to parasympathetic function, which may be treated in accordance with the subject invention. Examples of conditions that may be treated with the methods of the subject invention include, but are not limited to, cardiovascular diseases, e.g., atherosclerosis, coronary artery disease, hypertension, hyperlipidemia, cardiomyopathy, volume retention; neurodegenerative diseases, e.g., Alzheimer's disease, Pick's disease, dementia, delirium, Parkinson's disease, amyotrophic lateral sclerosis; neuroinflammatory diseases, e.g., viral meningitis, viral encephalitis, fungal meningitis, fungal encephalitis, multiple sclerosis, charcot joint; myasthenia gravis; orthopedic diseases, e.g., osteoarthritis, inflammatory arthritis, reflex sympathetic dystrophy, Paget's disease, osteoporosis; lymphoproliferative diseases, e.g., lymphoma, lymphoproliferative disease, Hodgkin's disease; autoimmune diseases, e.g., Graves disease, hashimoto's, takayasu's disease, kawasaki's diseases, arthritis, scleroderma, CREST syndrome, allergies, dermatitis, Henoch-schlonlein purpura, goodpasture syndrome, autoimmune thyroiditis, myasthenia gravis, Reiter's disease, lupus, rheumatoid arthritis; inflammatory and infectious diseases, e.g., sepsis, viral and fungal infections, wound healing, tuberculosis, infection, human immunodeficiency virus; pulmonary diseases, e.g., tachypnea, fibrotic diseases such as cystic fibrosis, interstitial lung disease, desquamative interstitial pneumonitis, non-specific interstitial pneumonitis, lymphocytic interstitial pneumonitis, usual interstitial pneumonitis, idiopathic pulmonary fibrosis; transplant related side effects such as rejection, transplant-related tachycardia, renal failure, typhlitis; transplant related bowel dysmotility, transplant-related hyperreninemia; sleep disorders, e.g., insomnia, obstructive sleep apnea, central sleep apnea; gastrointestinal disorders, e.g., hepatitis, xerostomia, bowel dysmotility, peptic ulcer disease, constipation, post-operative bowel dysmotility; inflammatory bowel disease; endocrine disorders, e.g., hypothyroidism, hyperglycemia, diabetes, obesity, syndrome X; cardiac rhythm disorders, e.g., sick sinus syndrome, bradycardia, tachycardia, QT interval prolongation arrhythmias, atrial arrhythmias, ventricular arrhythmias; genitourinary disorders, e.g., bladder dysfunction, renal failure, hyperreninemia, hepatorenal syndrome, renal tubular acidosis, erectile dysfunction; cancer; fibrosis; skin disorders, e.g., wrinkles, cutaneous vasculitis, psoriasis; aging associated diseases and conditions, e.g., shy dragers, multi-system atrophy, osteoporosis, age related inflammation conditions, degenerative disorders; autonomic dysregulation diseases; e.g., headaches, concussions, post

DOCUMENT-IDENTIFIER: US 20050214328 A1
TITLE: Methods of using and compositions comprising immunomodulatory compounds for the treatment and management of skin diseases or disorders

CLAIMS:

1. A method of treating, preventing or managing a skin disease or disorder characterized by overgrowth of the epidermis, keratosis, acne that does not comprise acne rosacea, or a wrinkle, which comprises administering to a patient in need of such treatment, prevention or management a therapeutically or prophylactically effective amount of an immunomodulatory compound, or a pharmaceutically acceptable salt, solvate, or stereoisomer thereof.

5. The method of claim 3, wherein the second active ingredient is a keratolytic, retinoid, anti-inflammatory agent, immunosuppressive agent, herbal product, antibiotic, collagen, botulinum toxin, interferon, or immunomodulatory agent.

6. The method of claim 3, wherein the second active ingredient is xanthium fruit, magnolia flower, 5-fluorouracil, masoprocol, trichloroacetic acid, salicylic acid, lactic acid, ammonium lactate, urea, isotretinoin, .alpha.-hydroxy acid, triamcinolone acetonide, collagen, botulinum toxin, or interferon.

19. The pharmaceutical composition of claim 18, wherein the second active ingredient is a keratolytic, retinoid, anti-inflammatory agent, antibiotic, collagen, botulinum toxin, interferon, herbal product, immunosuppressive agent or immunomodulatory agent.

20. The pharmaceutical composition of claim 19, wherein the second active ingredient is xanthium fruit, magnolia flower, 5-fluorouracil, masoprocol, trichloroacetic acid, salicylic acid, lactic acid, ammonium lactate, urea, isotretinoin, a-hydroxy acid, triamcinolone acetonide, collagen, botulinum toxin, or interferon.

22. A kit comprising: a pharmaceutical composition comprising an immunomodulatory compound, or a pharmaceutically acceptable salt, solvate, or stereoisomer thereof; and a pharmaceutical composition comprising a keratolytic, retinoid, anti-inflammatory agent, antibiotic, collagen, botulinum toxin, interferon, herbal product, immunosuppressive agent or immunomodulatory agent.

23. The kit of claim 22 comprising: a pharmaceutical composition comprising 3-(4-amino-1-oxo-1,3-dihydro-isoindol-2-yl)-piperidine-2,6-dio- ne or a pharmaceutically acceptable salt or solvate; and placental collagen or botulinum toxin.